

UNITED STATES NAVY

Medical News Letter

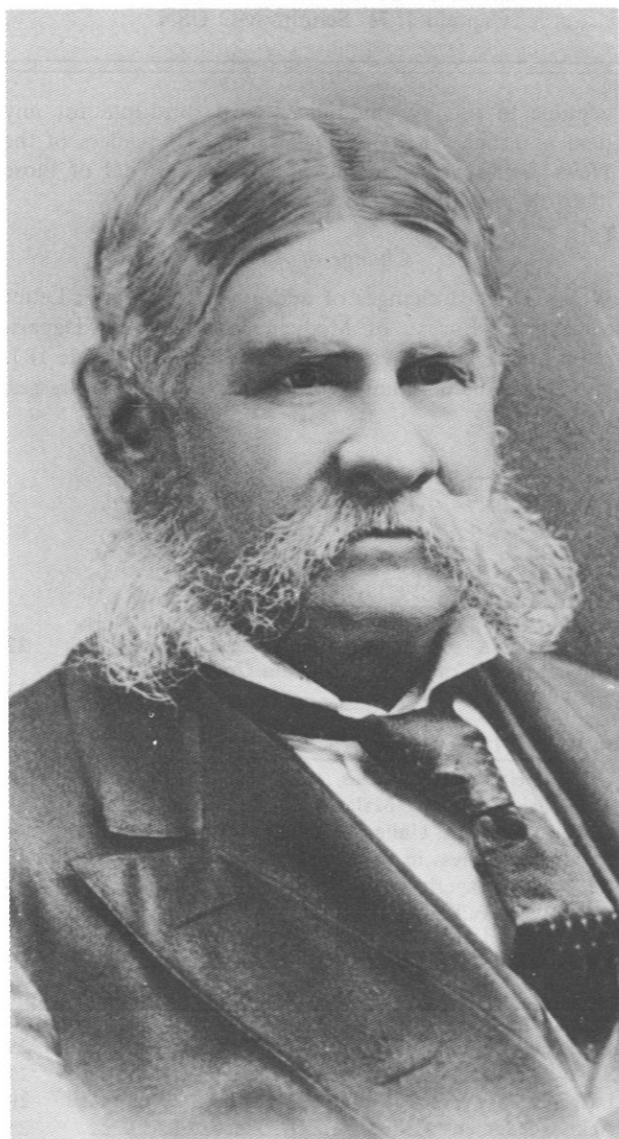
Vol. 49

Friday, 24 March 1967

No. 6

Surgeons General of the Past

(The eighth in a series of brief biographies)



The eighth Chief of the Bureau and fourth Surgeon General of the Navy, Joseph Beale, was born 30 December 1814 in Pennsylvania. He received his A.B. degree from the University of Pennsylvania in 1832, his A.M. in 1835, and his M.D. in 1836. Doctor Beale was appointed an assistant surgeon in the Navy September 6, 1837. His first sea duty was aboard the old sailing sloop John Adams in the East India Squadron from 1839-1841. After duty in the Home Squadron and the New York Naval Hospital, he served again on the John Adams which was engaged in suppressing the slave trade on the African coast, and was promoted to surgeon in 1848. During the Civil War he was attached to the steam sloop Susquehanna in the West Gulf Blockading Squadron from 1861 to 1863, participating in Admiral Farragut's attack on Forts Jackson and Phillip and the capture of New Orleans. After service at the Chelsea Naval Hospital from 1863 to 1865 he became Fleet Surgeon of the Asiatic Squadron. He was serving as President of the Board of Medical Examiners when he was appointed Surgeon General of the Navy in 8 July 1873. Medical Director Beale held this office until 2 February 1877. During his administration a volume of "Sanitary and Medical Reports" was published. Another volume, the last, was published later, the same type of material being gradually merged in the Annual Reports of the Surgeon General which had been initiated in the later sixties. A new hospital at Pensacola replacing the old one Admiral Farragut used in the Civil War, was constructed in 1875. It is interesting to note that in Doctor Beale's time a decade after the war and 10 years before the beginning of the steel-ship Navy, there were 156 officers in the U.S. Naval Medical Corps. Doctor Beale was placed on the retired list in 1877, and died in Philadelphia on 23 September 1889.

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MEDICAL NEWS LETTER

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The issuance of this publication approved by the Secretary of the Navy on 4 May 1964.

THREE-QUARTER HEPATECTOMY FOR TRAUMA:

REPORT OF A CASE

CAPT Joseph L. Whatley MC USN and CDR Richard R. Anderson MC USN**.*

With the advent of faster, (and more deadly!) means of transportation, both civilian and military physicians are seeing more patients with extensive penetrating or blunt abdominal trauma. In the past, the mortality and morbidity of patients with extensive hepatic injury was exceedingly high, particularly injury due to blunt trauma. Packing to control hemorrhage is unsatisfactory, since many patients exsanguinate after removal of the packs. Heavy catgut overlocking mattress sutures of the liver lacerations are also unsatisfactory—many continue to bleed; the development of hematomata, bile peritonitis, subphrenic abscesses and biliary fistulas is common.

Judd and Moore¹ reported an extensive resection for massive liver trauma in which they successfully resected the right lobe. They stated that to their knowledge at that time, their case was the second successful right lobectomy for trauma.^{1, 2}

The authors wish to report an even more extensive successful resection of hepatic substance, involving three of the four hepatic segments—the total right lobe and the medial segment of the left lobe.

D.E.C., a 21 year old Marine Private, was in an auto accident the evening of 6 August 1965. He was first treated at a civilian hospital, and transferred at 0345 on 7 August 1965 to USNH, Camp Pendleton. On arrival, he was in shock. Blood Pressure was 90/50, but the pulse rate was only 110. He complained of right upper quadrant pain and was confused and agitated. On palpation, there was diffuse tenderness and guarding of the entire abdomen, most marked in the right upper quadrant. Bowel sounds were hypo-active. Hematocrit was 28. An acute abdominal series and chest X-rays were considered to be within varied limits.

Because of persistent abdominal findings, a right lower quadrant paracentesis was done and gross blood was aspirated. He was taken to surgery immediately. In the operating room while he was being prepared for surgery, blood was started in the lower extremity veins. The abdomen was opened in the midline. About 1500 cc. free blood was found in the peritoneal cavity. There was an extensive stellate laceration of the liver, and large pieces of liver tissue were lying free in the right upper abdomen. The peritoneal attachments were completely severed except for the falciform ligament.

The midline incision was converted to a thoraco-abdominal and the diaphragm was split. The hepatoduodenal ligament was incised and the structures of the portal triad identified. The gallbladder was removed. Hemorrhage at this point was controlled by the assistant compressing the portal triad digitally. As dissection progressed and the assistant was needed elsewhere, a vascular clamp was applied to the triad, and released at 10-15 minute intervals. The right hepatic artery, bile duct, and portal vein were identified and doubly ligated with #20 silk and transected. It was noted at that time that the line of demarcation did not include all the damaged liver. The liver was further mobilized and the right hepatic vein identified, doubly ligated and transected.

At this point, bleeding was quite profuse. The middle and left hepatic veins could not be identified. Satinsky clamps were used to embrace the remaining damaged liver substance, and the damaged tissue was amputated. As the damaged liver was removed from anterior to posterior, the middle and left hepatic veins were identified, and the middle ligated and transected, the left preserved. This controlled the hemorrhage.

After blood replacement, the patient stabilized. The hilum was approached again, and the artery, bile duct and portal vein branches to the medial and

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The opinions or assertions contained herein are those of the authors and are not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large.

lateral segments of the left lobe were identified. The branches to the medial segment were ligated and transected, those to the lateral segment were preserved. The Satinsky clamps were removed one by one, and bleeders and leaking bile ducts were individually suture ligated.

The common duct was not drained. A large piece of gelfoam was placed over the raw surface of the remaining liver, and the omentum was sutured loosely to the capsule to enhance hemostasis. Four penrose drains were placed in the right hepatic fossa and brought out through a stab wound. Two other penrose drains were inserted in the region of the porta hepatis, and brought out through the incision, which was then closed.

The postoperative course was stormy. Treatment consisted of antibiotics (penicillin and streptomycin), whole blood, 10% Glucose and water, vitamin "K", balanced salt solution (Ringer's lactate), and human serum albumin. The first 48 hours postoperatively, the patient had marked hypoglycemia, hypoprothrombinemia and a reversed A/G ratio. He developed a massive right lower lobe atelectasis which gradually responded to intermittent positive pressure breathing. Subsequently, a subphrenic abscess developed which was drained on 17 September 1965. Initial cultures revealed aerobacter and proteus. Hemolytic staphylococcus aureus, coagulase positive grew in subsequent cultures. Multiple abscesses of the skin, abdomen and deltoid areas developed later. Large doses of Keflin were given and purulent drainage gradually became serous, but bile stained.

During the course of his severe illness, he lost approximately 42 pounds. By December 1965, however, he began to gain weight and was relatively asymptomatic except for a persistent biliary fistula. He was granted 30 days convalescent leave.

By March 1966, he had regained his normal weight, and liver function tests were all within normal limits. The biliary fistula persisted. He was transferred to a Veterans Administration Hospital near his home in August 1966, one year after his injury. At that time he was asymptomatic except for the biliary fistula. He was maintaining his original weight, appetite was good, and liver profile was normal. He undoubtedly will have to undergo further surgery to obliterate the biliary fistula.

I believe we learned or relearned several valuable lessons in treating this patient:

1. A relative bradycardia in a patient with abdominal injury, together with shock or borderline shock, should make one suspicious of severe liver

injury. Various authors have related this bradycardia to the absorption of bile, or to the effect on the heart of potassium released by damaged tissue.

2. With modern anesthetic and surgical techniques, and means for adequate blood replacement, extensive resection of damaged liver is possible. Quattlebaum and Quattlebaum³ have summarized the surgical anatomy of the liver.

3. Blood transfusions during the course of hepatic resection should be given in the upper extremities so that it may pass directly from the superior vena cava into the right atrium to maintain blood pressure and cardiac output. Blood given under pressure into the inferior vena cava causes brisk back-bleeding through the hepatic veins, furthering blood loss.

4. Initially, hemorrhage can be controlled by digital pressure over the portal triad, or by a non-crushing bulldog clamp, released intermittently every 10 to 15 minutes.

5. Satinsky non-crushing clamps are useful in those cases where vital structures are not readily identifiable, such as in this case the middle and left hepatic veins. Blood vessels and leaking bile duct radicals may then be individually suture ligated. The use of heavy interlocking mattress sutures should be avoided to decrease the amount of necrotic tissue.

6. Our patient developed a subphrenic abscess and a persistent biliary fistula. In retrospect, he might not have had these complications if some sort of common duct drainage had been resorted to at the time of surgery. Probably common duct drainage should be instituted in all extensive hepatic resections.

7. McDermott, et al⁴ studied the metabolic problems encountered postoperatively in patients following hepatic resection. Adequate blood replacement and prolonged, massive antibiotic therapy should be given to prevent infection and give the liver time to repair itself and resume its functions.

8. Carbohydrate metabolism: Initially, large amounts of glucose (10% G/W) should be given since the liver storage space for mobilizing glycogen has been greatly curtailed. After about 48-72 hours, there is a "rebound" phenomenon, and a tendency toward an abnormal glucose tolerance curve which persists for several weeks.

9. Protein metabolism: There is a reversal of the A/G ratio, which gradually returns to normal in about 6 months. The hepatic remnant appears unable to make sufficient albumin and serum albumin should be given for the first week postoperatively.

Otherwise, pulmonary edema and generalized anasarca may develop insidiously.

Prothrombin levels are consistently depressed despite additional vitamin K, but clinical evidence of clotting disorder was not encountered.

10. Lipid metabolism: No studies were made in this case. However, others⁴ report a gradual decrease in serum cholesterol and cholesterol esters the first week, and then a slow rise. Adipose tissue appears to be the primary source of fats following resection for about 4-6 months. Our patient lost 42 pounds before he began to regain his usual weight.

11. As long as the remaining segment of liver is normal, "ammonia intoxication" does not appear to be a grave problem. "Balanced salt" (Ringer's

lactate solution) should be given during and after surgery. Two parts normal saline and one part Ringer's lactate approximates normal extracellular fluid.

Summary

A successful case of three-quarter hepatectomy for trauma is reported, together with observations on the operative and postoperative management of these cases.

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HUMAN EXPERIMENTATION IN INFECTIOUS DISEASES

Harry F. Dowling MD, JAMA 198(9):133-135, November 28, 1966.

Rarely are investigators so bold as to produce disease in man, but in the field of infectious diseases they have done so repeatedly. Often these diseases were mild, usually they were self-limited, and sometimes they could be cured with drugs; yet the subject of the experiment always ran some risk of disability or death. Since exposing men to such hazards contrasts sharply with the concepts of human dignity embodied in Judeo-Christian ethics, it is interesting to see how these concepts affected the evolution of human experimentation.

The surge of scientific discovery during the Renaissance and early modern period involved mostly inanimate objects at first and later animals. The thread of experimentation on humans can be picked up easily in 1721 when a smallpox epidemic was raging in London. George I, loath to have his grandchildren inoculated as recommended by Lady Mary Montagu, ordered the virus given first to six condemned criminals and then to seven orphans. Such trials and the successful inoculation of hundreds of people with smallpox made it easier for Jenner on May 14, 1796, to inoculate an 8-year old boy with material from a cowpox pustule and then, "in order to ascertain whether the boy was secure from the contagion of the smallpox, he was inoculated the first of July following with variolous

matter, but no sensible effect was produced on the constitution." Thus, Jenner verified his hypothesis by a human experiment. Later he inoculated other children and adults, passed vaccinia from child to child, and challenged with smallpox the first and fifth child in a series; he found that vaccination consistently protected against smallpox.

Other early experiments included the transmission of measles in 1758, gonorrhea in 1767, and plague in 1781. In the 19th century, syphilis was transmitted from a primary lesion, malaria by injection of blood, and yellow fever by the bite of mosquitoes.

The discoveries of Pasteur and Koch accelerated human experimentation in infectious diseases and made the results more reliable by providing pure cultures of microorganisms. In 1892 von Pettenkofer, trying to prove to the world that he was right in his belief that vibrios could not cause cholera, swallowed a fresh culture. Fortunately, only a light diarrhea developed. Shiga's experience was more uncomfortable. After he injected "one half an agar culture" of dysentery bacilli under his skin, headache, chills, fever, and muscular aches developed, followed by a localized abscess.

Metchnikoff's mercurial temperament led him to perpetuate two dangerous experiments on himself. At the University of Odessa in 1881, discouraged with political interference in the university, he swallowed a culture of *Borrelia*. He became severely

From the Department of Medicine, University of Illinois, Chicago.
Presidential address read before the Infectious Diseases Society of America, Philadelphia, Oct 24, 1966.

ill but lived to drink a culture of vibrios several years later, this time without any effect. Self-experimentation was becoming the vogue. In 1885 Garre produced an abscess by rubbing staphylococci on the skin of his arm.

Thus, for the most part, the early experiments were unsystematic and poorly planned, occasionally performed on the spur of the moment, and frequently dangerous. Some deaths occurred; Cerutti inoculated six persons with plague pus and five died. Many other such incidents were not reported. Walter Reed's experiments on yellow fever marked not only the turn of the century but also a change in attitude. They were carefully planned and comprehensive, testing sequentially the spread of the disease by mosquitoes, by the air, and by fomites. The volunteers were true volunteers and arrangements had been made beforehand to nurse the victims of the disease back to health.

Most 20th century experiments in volunteers followed the same pattern, beginning with the transmission of plague in 1906, amebiasis in 1913, *Hemophilus influenzae* infections in 1936, *Mycoplasma* (primary atypical) pneumonia in 1945, tularemia in 1961, and typhoid in 1963. Among viral infections, the common cold was the first given to volunteers, in 1914, later, influenza, rubella, and infectious and serum hepatitis were given. Viruses grown in tissue culture were first used to produce coxsackievirus and adenovirus infections in 1950. Since then, volunteers have been infected with parainfluenza, echovirus, respiratory syncytial, rubella, and sandfly fever viruses and rhinoviruses.

Parallel with refinement in laboratory techniques, experiments became more elaborate and more comprehensive. Investigations on the chemotherapy of malaria employed 4,400 prisoners. In Salisbury, England, more than 3,500 vacationing and honeymooning couples have been isolated for ten days while taking their chances on a common cold developing. Nearly 4,000 student volunteers have participated in experiments on respiratory infections by our group at the University of Illinois, and approximately 800 prisoners have participated in similar experiments at the National Institutes of Health. No deaths or serious complications have occurred in any of these studies.

Besides being more complex and more extensive, recent studies have asked more questions than previous ones: Does chilling predispose to the common cold? What drugs will prevent or cure malaria or influenza? How large must particles be to carry

viruses into the lungs? What physiological changes occur in severe infections?

These changes in human experimentation derive from the accumulation of knowledge and improvements in scientific techniques. But sophistication has not been confined to the laboratory. In recent years, experiments on humans have been planned more carefully and have been better controlled. Statistical methods are commonly used in their design and interpretation. More important would be an improvement in our attitude toward the use of humans as experimental subjects. Information on this subject is scant, but there is some.

At the time of George I, since criminals were treated as depraved persons, tamable only by the whip and often condemned to death for trivial offenses, orders from a king to give them smallpox virus were not considered amiss. Indeed the rarity of experimentation upon criminals resulted from neither lofty ethnics nor a dearth of subjects but rather from lack of interest and want of imagination on the part of the medical profession. In contrast, in the early 20th century, when Walker and Sellards used Filipino prisoners for experiments on amebiasis, the prisoners signed an agreement to participate without coercion or even a promise of pardon. During World War II, the deployment of our troops in tropical areas, the high morbidity rates from malaria, and the unavailability of quinine made the search for synthetic drugs imperative. Since these had to be tested in humans and since manpower was in short supply, it was natural to turn to the large pools of men in prisons. After the war, the despicable actions of Nazi physicians in subjecting prisoners to crippling and fatal experiments came to light, and investigations of these crimes caused us to rethink and restate our ethics on the use of prisoners as subjects. Present codes require that animals be used beforehand to achieve as much information as possible, that the subject give his informed consent, that his health and life be safeguarded during and after the experiment, and that he be allowed to withdraw at any time.

In recent years a number of experiments have been carried out on prisoners. The lay press seems to approve. The *American Mercury* (Dec 5, 1954) spoke favorably of experiments with humans on syphilis and the *New York Times* (Dec 4, 1960) thought volunteer experiments gave the criminal a feeling of usefulness and an opportunity for atonement. One prisoner told the reporter, "It's the only decent thing I ever did."

The greatest potential pool of experimental subjects in the past was composed of persons disadvantaged because of race, class, or economic status. One of the children inoculated by Jenner lived in a poorhouse. In recent years, protests against the use of the poor have been heard; for instance the severe criticism in *The Nation* (June 29, 1921) of experiments in which scurvy was produced in infants in an orphans home. "A child is placed in an infant asylum," the author said, "because the parents are too destitute to care for it properly. It is never intended to take the place of a guinea pig in a dietetic laboratory."

In a country said to be ruled by its children, it is hard for us to realize that children had few rights only a short time ago. In the latter half of the 18th century, for instance, spinning machines were so simplified by the inventions of Arkwright and others that children could run them. At the ages of 5 or 6 years they were brought from the poorhouses of Edinburgh and forced to work for long hours in the "dark, Satanic mills." Not until a century later was much done about it.

Perhaps Jenner used so many children because most adults had had smallpox, but one wonders whether he had the consent of parents. He does not say, although one suspects he didn't when he writes, "my nephew, Mr. Henry Jenner, at my request inserted the vaccine virus into the arm of a child about twenty hours old." One also wonders at Benjamin Waterhouse's action in sending his recently vaccinated son to the smallpox hospital to be inoculated so as to prove that his opinions on vaccination were correct.

The trials of the Nazi war criminals focused our attention on the meaning of consent. In other words, when is a volunteer not a volunteer? Some of the earlier experimental subjects patently were not. In 1721, the same year that George I ordered that smallpox be given to six criminals, Dr. Zabdiel Boylston inoculated two Negro slaves and his own son. It was not recorded that he asked the permission of any of them. Eighty years later, while the controversy over vaccination was still raging, we read that "two members of the Board of Health . . . offered their own sons for the test," while in the Foundling Hospital in Naples children were vaccinated and then put to bed with smallpox patients.

Other inoculations are equally suspect. Bockhart in 1883 gave gonorrhea to a man with dementia praecox. Ricord around 1850 repeatedly transferred pus from the primary lesion of syphilis to other sites on the patient's body, and Piringer in 1841 saturated

pieces of linen with pus from infected eyes and gave them to patients as eye-wipers—infections developed in those who used recently contaminated cloths.

A writer in the *Catholic World* (January 1900) called such experiments "murder in the name of science" and quoted a Swedish doctor as saying,

"I should perhaps have chosen animals . . . But the most fit subjects, calves, were obtainable only at considerable cost. There was, besides, the cost of their upkeep so I concluded to make my experiments upon the children of the Foundling's Home and obtained kind permission to do so from the Head Physician . . ."

The experiments of Walter Reed stand in sharp contrast. The volunteers were true volunteers; there was no coercion. Although compensation had been set at \$250 for each volunteer, at least two, Kissinger and Moran, refused to accept anything. At one point, when Reed was about to take part in an experiment himself because he was one subject short, Private John H. Andrus offered himself instead. Only after considerable argument were Reed's colleagues able to persuade him to accept the substitute. A severe case of yellow fever developed, which impelled Reed to write to Surgeon General Sternberg: "I am very uneasy about the nonimmune soldier (Andrus) who got the same quantity of blood . . . Should he die I shall regret that I ever undertook this work. The responsibility for the life of a human being weighs upon me very heavily just at present, and I am dreadfully melancholic."

Present-day investigators have been equally solicitous of the volunteers' welfare. Knight has described the precautions taken at the National Institutes of Health; others have used similar methods. Volunteers have usually been students, criminals, or conscientious objectors to military service. Of the latter, Tigertt wrote,

"It is a privilege to acknowledge the unselfish cooperation of the large volunteer group of Seventh Day Adventist soldiers whose participation is making these studies possible. They have not only served as manifest volunteers, but selected, nonvaccinated, nonimmune personnel have been intimately associated with the exposed groups. . . ."

Both *Time* (Sept 27, 1954) and the *Reader's Digest* (January, 1963) extolled the conduct of conscientious objectors as volunteers.

In fact, groups outside the medical profession now frequently participate in the discussion of human experimentation and criticize not only the methods of obtaining volunteers but the scientific

justification of the experiments. The *New York Times* (Aug 20, 1901) asked why, after the original experiments of Walter Reed which were "obviously of such priceless value," the chief surgeon had not "long ago forbidden duplication of perilous experiments that could not possibly teach anything new" before the death of one of the volunteers. Likewise, an article in the *Saturday Review* (Feb 5, 1966) recently criticized an experiment in which cancer cells were injected under the skin.

Alfred North Whitehead once said, "I am struck by the fact that mankind has not advanced morally, to speak of, for the past two thousand years." Perhaps not, but the records of human experiments over the past few centuries show more concern by

the medical profession for the rights and welfare of volunteers, more care in the planning and conduct of human experiments, less exploitation of disadvantaged groups, many attempts to define the meaning of informed consent and more public discussion of the facts. Perhaps this is a small step in human progress after all.

The experiments on the common cold at the University of Illinois were supported by research contracts DA-49-007-MD-421 and DA-49-193-MD-2410 from the US Army Medical Research and Development Command.

(The references may be seen in the original article.)

PERSISTENT PROTEINURIA IN ASYMPTOMATIC INDIVIDUALS: RENAL BIOPSY STUDIES ON 50 PATIENTS†

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Milit Med 131(10):1311-1317, October 1966.

Persistent proteinuria is generally considered *prima facie* evidence of renal disease in the absence of lower urinary tract pathology. However, few studies are available on large numbers of asymptomatic healthy appearing patients with persistent proteinuria. The need for complete studies on these individuals is well known to military physicians who are frequently faced with such patients discovered on routine periodic physical examinations.

Methods and Materials

This study comprises 50 individuals found to have asymptomatic persistent proteinuria on routine examinations. All patients were studied in detail at the USAF Hospital Travis or at Wilford Hall Hospital at Lackland Air Force Base, Texas.

A simple test previously outlined was used to screen orthostatic protein excretion patterns from persistent ones. In brief, this is a qualitative test requiring overnight supine positioning with ex-

amination of the urine before arising and after ordinary ward activity. Patients with urine samples showing proteinuria in both upright and supine positions were classified "persistent." Only those patients with proteinuria present in urine samples taken at least 30 days apart were studied. Patients with previously diagnosed glomerulonephritis, pyelonephritis, or systemic diseases known to affect the kidneys were excluded. J.E.S. was included because this young man's proteinuria preceded the onset of clinical gout by seven years. W.J.S. had a history of "possible nephritis" as a small child, however several routine urinalyses had been normal prior to the appearance of asymptomatic proteinuria. Those patients with elevated blood pressures at the time of our study had all been observed to have proteinuria several months to several years prior to abnormal blood pressure recordings. All patients had normal renal function as measured by endogenous creatinine clearance of greater than 80 ml/min., blood urea nitrogen of less than 20 mg. %, urine specific gravity above 1.018, and prompt bilateral excretion of dye on intravenous pyelogram study. One of the authors examined a urinary sediment from each patient, and a mid-stream urine culture was obtained.

†The views expressed in this manuscript are the personal opinions of the authors, and are not to be construed as a statement of official U.S. Air Force policy.

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Percutaneous renal biopsies were performed on all but one patient, and were all uncomplicated. F.C.E. had an open surgical biopsy. Tissue samples were immediately placed in 10% neutral-buffered formalin solution. Sections were cut from paraffin blocks with a conventional microtome. Sections ranged between 3 microns and 6 microns in thickness. All slides were reviewed by at least three examiners using the standard light microscope. All tissue specimens included in the report had at least four glomeruli and most had greater than ten. Periodic acid-Schiff and hematoxylin and eosin stains were made on all. Selected cases had stains for amyloid as well.

Three general classifications were given: (1) Normal diagnoses were agreed upon by all three examiners; (2) Probably abnormal was used to describe those cases where at least two examiners felt there were significant minor glomerular abnormalities (focal hypercellularity or capillary loop thickening)

but unanimous opinion could not be reached; (3) Definitely abnormal cases were then classified descriptively. Membranous glomerulonephritis was used to describe those cases of glomerular disease showing thickened glomerular capillary loops without appreciable increase in cellularity. Proliferative glomerulonephritis was used to classify cases of glomerular pathology showing an increase in glomerular cellularity without significant capillary loop thickening. Mixed glomerulonephritis was used to describe the histology of cases showing both glomerular capillary loop thickening and hypercellularity. No attempt was made to infer an etiology with this descriptive classification of glomerular disease.

Results

Table I lists pertinent information on each patient and the histologic diagnoses.

TABLE I

Patient	Age	Sex	Duration	History-Physical-Laboratory	Biopsy Diagnosis
J.E.S.	34	M	8 yr	Acute gouty arthritis 1961 RBC's 8-12/HPF in urine sediment.	Focal areas of interstitial scarring with chronic inflammatory reaction. Gouty nephropathy?
W.D.C.	30	M	7 yr	1.7 gm/24 hr. BP 186-120/102-86	LIPOPHAGIES in interstitium. Bands of interstitial inflammation with mild glomerular scarring.
F.C.B.	47	M	4 mo	1.1 gm/24 hr. BP 148/90	Mild arteriolar nephrosclerosis.
E.R.S.	45	M	6 yr	—	5 out of 23 glomeruli hyalinized. Others normal.
O. McF.	42	M	9 yr	2-6 gm/24 hr. BP 150/100	Moderate proliferative glomerulonephritis.
D.S.	32	M	4 mo	1.2 gm/24 hr	Moderate proliferative glomerulonephritis.
W.J.S.	29	M	3 mo	1.0 gm/24 hr. "Nephritis" as a child.	Mild proliferative glomerulonephritis.
J.D.O.	25	M	14 mo	0.8 gm/24 hr	Mild proliferative glomerulonephritis.
O.W.	22	M	6 mo	—	Mild proliferative glomerulonephritis.
A.T.B.	17	M	2 mo	0.6 gm/24 hr	Mild proliferative glomerulonephritis.
P.J.K.	24	M	1 yr	0.8 gm/24 hr	Mild proliferative glomerulonephritis.
L.R.E.	26	M	3 mo	0.58 gm/24 hr	Mild proliferative glomerulonephritis.
F.C.E.	35	M	6 yr	0.85 gm/24 hr. IVP showed defect in right upper calyx. Open surgical biopsy—fetal lobulation.	Mild proliferative glomerulonephritis.

TABLE I—(Continued)

Patient	Age	Sex	Duration	History-Physical-Laboratory	Biopsy Diagnosis
J.A.L.	27	M	5 yr	2.1 gm/24 hr. BP 160/100–120/84.	(Biopsy 1962—Minimal proliferative glomerulonephritis Normal?). Biopsy 1963—Mild proliferative glomerulonephritis.
E.C.F.	21	M	11 mo	1.58 gm/24 hr	Moderate membranous glomerulonephritis.
S.B.L.	54	F	7 yr	1.23 gm/24 hr	Moderate membranous glomerulonephritis.
E.L.T.	35	M	10 yr	4.1 gm/24 hr. Allergic to Penicillin. Mother diabetic. BP 170–126/100–86	Moderate membranous glomerulonephritis with focal interstitial inflammation.
S.E.B.	34	M	16 yr	1.9 gm/24 hr. BP 164–110/100–70 Hmct. 47%, Hbg. 16.4 gm. %	Mild focal membranous glomerulonephritis.
R.E.G.	25	M	3 yr	Dark urine in 1959. Epilepsy in early childhood controlled with Phenobarbital.	Mild membranous glomerulonephritis.
J.F.M.	24	M	1 yr	3 gm/24 hr	Mild membranous glomerulonephritis.
G.C.	39	M	2 yr	3.9 gm/24 hr. BP 150–110/124–84	Mild membranous glomerulonephritis.
E.B.B.	39	M	6 yr	1.4 gm/24 hr. Nocturia×2 for 10 yr BP 140/90. Orthostatic proteinuria at age 33.	Mild membranous glomerulonephritis (1960 and 1962).
N.R.W.	38	M	6 yr	BP 150–128/94–86	Normal in 1962. Mild membranous glomerulonephritis—1965.
R.S.N.	51	M	26 yr	BP 150/104	Mild membranous glomerulonephritis and mild arteriolar nephrosclerosis.
G.C.C.	38	M	13 yr	1.8 gm/24 hr	Moderate mixed glomerulonephritis.*
M.E.M.	22	M	2 yr	1.2 gm/24 hr	Mild proliferative glomerulonephritis (1960). Mild to moderate glomerulonephritis (1962).
H.D.H.	39	M	3 mo	EKG showed old diaphragmatic infarction. BP 150/100. Urine culture —(1) E. Coli (2) Negative.	Mild mixed glomerulonephritis.
H.H.M.	52	M	3 yr	3.3 gm/24 hr. Duodenal ulcer for 10 years.	Mild mixed glomerulonephritis.
F.S.C.	23	M	6 yr	1.2 gm/24 hr	Mild mixed glomerulonephritis.
D.S.G.	27	M	2 yr	0.73 gm/24 hr	Mild mixed glomerulonephritis.

* Mixed Glomerulonephritis—i.e. Membranous and Proliferative Glomerulonephritis.

TABLE I—(Continued)

Patient	Age	Sex	Duration	History-Physical-Laboratory	Biopsy Diagnosis
W.C.N.	42	M	5 yr	0.3 gm/24 hr BP 150/90	Mild focal mixed glomerulonephritis.
C.D.C.	32	M	2 mo	BP 134-120/96-84 Hmct. 59%, Hbd. 18.5 gm. % CR. 51, RCV=35 mc/kg (normal 23-33), I-131 RCV-44 ml/kg (normal 26-42)	Mild focal mixed glomerulonephritis.
L.C.C.	41	M	6 mo	1.9 gm/24 hr BP 192-130/118-90	Mild mixed glomerulonephritis with focal interstitial inflammation.
A.J.B.	26	M	4 yr	0.33 gm/24 hr. Albuminuria orthostatic in 1960.	Mild mixed glomerulonephritis.
E.D.	23	F	7 mo	1.4 gm/24 hr Hmct. 33%, Hbg. 10.6 gm. %. Onset during 7th month of pregnancy.	Mild mixed glomerulonephritis.
S.S.D.	20	F	2 mo	0.8 gm/24 hr. Cleared about 3 mo. later.	Minimal mixed glomerulonephritis.
M.L.G.	47	M	4 yr	2.1 gm/24 hr. BP 164-126/104-80. Ulnar neuropathy.	Probably abnormal. Minimal proliferative glomerulonephritis.
W.T.S.	21	M	1 mo	—	Probably abnormal. Minimal focal proliferative glomerulonephritis.
R.W.H.	40	M	5 yr	1.2 gm/24 hr. BP 168/110. Labile hypertension 10 yrs.	Probably abnormal. Minimal focal membranous glomerulonephritis.
C.V.J.	23	M	1 mo	BP 130/90	Probably abnormal. Minimal proliferative glomerulonephritis.
B.K.M.	27	M	8 yr	1.3 gm/24 hr	Probably abnormal. Minimal proliferative glomerulonephritis.
J.B.B.	38	M	1 yr	Erythrocyte sedimentation rate=25 mm/hr. (Wintrobe)	Probably abnormal. Focal membranous glomerulonephritis.
T.U.R.	29	M	8 yr	2.86 gm/24 hr	Probably abnormal. Minimal focal mixed glomerulonephritis.
J.A.S.	16	F	6 yr	2.4 to 6 gm/24 hr. Frequent otitis media.	Normal.
M.K.E.	30	F	3 mo	0.16 gm/24 hr	Normal.
S.W.B.	35	M	3 mo	1.8 gm/24 hr	Normal.
R.A.G.	32	M	9 yr	0.5 gm/24 hr. Proteinuria was orthostatic at age 27. Had polyarthritis at age 14.	Normal.
J.W.W.	31	M	6 yr	Treated for prostatitis on two occasions.	Normal.
B.D.D.	32	M	6 mo	0.6 gm/24 hr. IVP-minor calyceal blunting? BP 184/112.	Normal.
E.W.S.	21	M	1 mo	1.8 gm/24 hr	Normal.

A diagnosis of glomerulonephritis was made on the renal biopsies from thirty-two patients. An additional seven patients had nonspecific focal glomerular lesions consisting of hypercellularity or capillary loop thickening found on renal sections. Thus, 78% had glomerular abnormalities. The renal biopsies of two patients showed an interstitial nephritis. One biopsy revealed mild arteriolar nephrosclerosis and seven patients' renal biopsies were considered normal. The biopsy listed as unclassified had five hyalinized glomeruli out of twenty-three without interstitial inflammation or periglomerular fibrosis. The remaining glomeruli appeared normal.

Three patients in our group had previously had well documented orthostatic proteinuria. In one, our biopsy was normal. In one, there was mild to moderate diffuse membranous glomerulonephritis. The third patient's biopsy showed diffuse membranous and proliferative glomerulonephritis. Recent studies on young patients with fixed and reproducible orthostatic proteinuria have shown a significant percentage to have abnormal renal biopsies.

In four patients serial renal biopsies were available for study. E.B.B., had renal biopsies in 1960 and 1962 which showed mild membranous glomerulonephritis on both occasions. J.A.L., was found to have minimal evidence of proliferative glomerulonephritis in 1962. Our biopsy in 1963 revealed definite mild proliferative glomerulonephritis. M.E.M., had a proliferative glomerulonephritis on biopsy in 1960. In 1962 renal biopsy showed a mild to moderate mixed glomerulonephritis. A normal renal biopsy was obtained from N.R.W. in 1962. A second biopsy in 1965 showed membranous glomerulonephritis. Thus, three of four patients in whom serial renal biopsies were available had histologic evidence of progression of their renal disease which was completely unsuspected on the basis of normal renal function tests.

In C.C., several routine hematocrits had been elevated in the range of 60%. Blood volume determinations using Cr revealed a total blood volume of 59 ml/kg (normal 55-78). The red cell volume was 35 ml/kg (normal 23-33) and the plasma volume was 24 ml/kg (normal 32-45). This modest increase in circulating red cell volume is felt to be another example of secondary polycythemia in association with renal disease. The renal biopsy in this patient showed a mixed glomerulonephritis. Previous reports have shown that seemingly mild

renal parenchymal disease may be associated with secondary polycythemia.

Urinary sediment examination was uniformly of no diagnostic help as most patients had occasional hyaline and/or granular casts. In only one instance were significant blood cell elements present in the urine sediment. This was in J.E.S. who had 8-12 RBC/HPF and was known to have gouty arthritis at the time of the biopsy.

The degree of proteinuria was also not helpful in making a diagnosis. Most spilled about 1 gm/24 hr. The maximum was 6 gm/24 hr. in J.A.S. whose biopsy revealed normal renal tissue.

The duration of the proteinuria ranged from a minimum of one month to a maximum of 26 years. S.S.D. had a two month history of persistent proteinuria when she was biopsied. The histologic diagnosis was mild proliferative glomerulonephritis. Approximately two months later follow-up revealed complete clearing of the proteinuria. R.S.N. a 51 year old male had well documented asymptomatic proteinuria for 26 years. Mild hypertension appeared after 25 years and our biopsy showed mild membranous glomerulonephritis and early arteriolar nephrosclerosis.

The intravenous pyelogram was abnormal in only two studies. In one a defect in the upper calyx on one side led to exploratory laparotomy where a fetal lobulation was discovered. A biopsy was obtained and revealed proliferative glomerulonephritis. A second patient had questionable blunting of the minor calyceal system.

Seventeen patients had diastolic blood pressure elevations at the time we studied them. Table II lists the tissue diagnoses in this group of patients. Ten had sufficient elevations of their diastolic blood pressure that drug therapy was started. Out of 22 patients with a well documented history of proteinuria for at least five years, 11 were hypertensive.

TABLE NO. II
HISTOLOGIC DIAGNOSIS ON HYPERTENSIVE PATIENTS

Membranous Glomerulonephritis	8
Proliferative Glomerulonephritis	3
Mixed Glomerulonephritis	3
Normal	1
Arteriolar nephrosclerosis	1
Interstitial Nephritis	1
	—
Total	17

These results are in complete agreement with Dr. S. E. King's report of a high incidence of hypertension in patients with persistent proteinuria followed over five years.

DISCUSSION

We elected not to use pyelonephritis as a histologic diagnosis because of the uncertainties and difficulties involved. The two cases of interstitial nephritis might well be considered as pyelonephritis on the basis of interstitial round cell infiltrates and periglomerular fibrosis. Neither had any clinical or laboratory evidence of renal parenchymal infection. In one there were focal areas of interstitial inflammation and fibrosis extending from cortex to medulla in the biopsy specimen. This man had well documented hyperuricemia and gouty arthritis. Since gouty patients have been shown to have interstitial inflammation secondary to urate deposits, it seems possible that this man's kidney disease was related to gout and infection need not have played a part. The second case of interstitial nephritis had numerous clusters of foam cells interstitially in the biopsy. None of the known diseases associated with renal foam cells were present in this patient. Only one of our patients had a positive urine culture. This man's renal biopsy showed glomerulonephritis and his repeat urine culture was negative. Two patients had focal interstitial inflammatory reaction in addition to well defined diffuse glomerular disease. Thus, including the patient with the questionable calyceal blunting on intravenous pyelogram, there was some evidence to suggest pyelonephritis could have played a part in the renal disease of six patients.

The normal renal biopsies obtained in this group deserve further comment. We suspect some of these people have glomerular disease not discernible by light microscopy. Support for this conjecture seems well demonstrated by our experience with N.R.W. who had a normal renal biopsy by light microscopy in 1962 and definite glomerular disease three years later when rebiopsied. Another explanation in keeping with the concept that persistent proteinuria is always due to renal disease would be the presence of focal renal disease not sampled by the biopsy needle.

These observations certainly confirm the relationship between persistent proteinuria and underlying renal disease. They also demonstrate that some cases show histologic progression of the underlying renal disease that is not appreciated unless renal biopsies are performed serially. The prognosis for the individual patient with persistent proteinuria remains

uncertain. Perhaps long term follow-up studies utilizing renal biopsies will help clarify this problem.

The finding of normal renal function tests, a non-diagnostic urinary sediment, and an otherwise healthy appearing patient should not deter the physician from making a diagnosis of renal parenchymal disease in the presence of persistent proteinuria. Percutaneous renal biopsy is a safe method of making a histologic diagnosis and in selected cases offers significant information to help with prognosis when performed serially.

Summary

Renal biopsies from 50 individuals with asymptomatic persistent proteinuria are discussed. Seventy-four percent had definitely abnormal renal tissue. An additional 12% had minor focal glomerular changes that were probably abnormal. Only 14% had normal renal biopsies. By far the most frequent histologic diagnosis was glomerulonephritis. Evidence of renal parenchymal infection was present in a small number of patients. Renal biopsy offers the only method of making an etiological diagnosis in most of these patients.

Editor's Note—A similar study was begun in June 1959 at the Lackland Air Force Base, Texas, and Durham, North Carolina. Fifty-seven of sixty-four young men who had exhibited "fixed and reproducible" orthostatic proteinuria in 1959 were evaluated after five years. The investigators (MAJ F. R. Lecocq MC USAF, MAJ J. J. McPhaul MC USAF, and R. R. Robinson M.D., Fixed and Reproducible Orthostatic Proteinuria, V, Results of a 5-year Follow-up Evaluation, *Annals of Internal Medicine* 64:557-569, March 1966) concluded that in their opinion "fixed" orthostatic proteinuria is not always a transient condition of adolescence and that it may well reflect an early form of parenchymal renal disease whose final clinical expression is still unknown. The following are references to this study during the five year period:

9. Robinson, R. R., Glover, S. N., Phillippi, P. J., Lecocq, F. R., Langelier, P. R.: Fixed and reproducible orthostatic proteinuria. I. Light microscopic studies of the kidney. *Amer. J. Path.* 39:291, 1961.

10. Robinson, R. R., Ashworth, C. T., Glover, S. N., Phillippi, P. J., Lecocq, F. R., Langelier, P. R.: Fixed and reproducible orthostatic proteinuria. II. Electron microscopy of renal biopsy specimens from 5 cases. *Ibid.*, p. 405.

The figures and bibliography not reproduced may be seen in the original article.

11. Robinson, R. R., Lecocq, F. R., Phillippi, P. J., Glenn, W. G.: Fixed and reproducible orthostatic proteinuria. III. Effect of induced renal hemodynamic alterations upon urinary protein excretion. *J. Clin. Invest.* 42:100, 1963.

12. Robinson, R. R., Glenn, W. G.: Fixed and reproducible orthostatic proteinuria. IV. Urinary albumin excretion by healthy human subjects in the recumbent and upright postures. *J. Lab. Clin. Med.* 64:717, 1964.

FATAL BLEEDING ULCER

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Avery Jones has described the fallacies inherent in most published statistics concerning management of upper gastrointestinal hemorrhage. Because of many variables, it is not possible critically to compare treatment regimens from different centers. Nor does it seem fruitful to attempt to "standardize" clinical material using laboratory data, for this in no way reflects the importance of such factors as age, associated diseases and the underlying gastroduodenal disorder. In addition, the concept of functional blood volume challenges the value of conventional blood volume determinations. There is no objective method of measuring the patient's ability to tolerate and compensate for hemorrhage, yet this ability largely determines survival.

It is not satisfactory to compare different regimens applied in one hospital in consecutive but nonetheless different eras, for improved results always follow enthusiastic interest in a subject, and adjuvant therapy is bound to progress over the time span. Sizable concomitant clinical trials have not been conducted, presumably because of excessive time needed to observe sufficient patients.

To study the factors associated with fatality the charts of 50 consecutive fatal cases of bleeding peptic ulcer seen at the Ottawa Civic Hospital over a 9-year period were reviewed in detail.

Review of Clinical Observations

History of Hemorrhage. About half the deaths occurred during the first hospital admission for ulcer and in three-fourths during the first admission for gastrointestinal bleeding. Thirty-nine of the 50 patients had previously been diagnosed as having peptic ulcer (Table 1).

It is important to realize that of the 38 patients admitted because of hemorrhage, 24 gave a history suggestive of bleeding in the week prior to the episode that resulted in admission. Thus the admission hemorrhage was often the second or third of the current series of hemorrhages.

TABLE 1. INCIDENCE OF HEMORRHAGE AND KNOWLEDGE OF ULCER IN 50 CASES

<i>Past History</i>		
Previous admission for ulcer	26	
Hemorrhage		14
Other manifestation		12
No previous admission	24	
Known ulcer		13
Dyspepsia, unproved etiology		6
No dyspepsia or ulcer		5
<i>Reason for present admission</i>		
Cause other than ulcer	8	
Ulcer, hemorrhage	38	
Ulcer, other manifestation	4	

Nature of Hemorrhagic Episodes. The authors believe that insufficient attention has been given to Avery Jones' observation that bleeding usually occurs in short, sharp, self-limited bouts that seldom exceed 30 minutes in duration. He and others have pointed out that at emergency operation bleeding has usually stopped, and recurs only if the ulcer is disturbed. Considering the violence of bleeding that follows and the size of vessels usually involved, it is obvious that bleeding could not continue for long without the patient exsanguinating. Yet, as can be

seen in Table 2, this rarely occurs. The bleeding must therefore, stop spontaneously. We often forget that because of gastric stasis, vomiting of blood may continue some time after bleeding from the ulcer has stopped. Persisting hypotension may indicate persisting decompensation but not necessarily continu-

ing blood loss. The decreasing hematocrit from hemodilution only records events of 12 to 48 hours earlier. Many reports describe the numerous self-limiting hemorrhages that occur before death from an aortic aneurysm which ruptures into the gastrointestinal tract.

TABLE 2. RECURRENT HEMORRHAGE IN HOSPITAL

Recurrence	0	1	2	3	4	5	6	7	8	9	10
No. Patients	5	7	6	10	7	6	5	1	1	1	1

Median number of hemorrhages in hospital: 3, Median day of death: 6th.

The number and nature of bleeding episodes were determined by reviewing recorded details concerning the occurrence, nature, timing, and size of the hematemeses; the occurrence of incontinent or frequent liquid tarry stools or bloody stools; the occurrence of shock, hypotension, weak or rapid pulse, restlessness, apprehension or sweating, and the response of these and hemoglobin levels to transfusion. Eighty percent of bleeding episodes were abrupt, vigorous and short-lived. In most of the remainder, continued slower bleeding was suggested by repeated small hematemeses or small often incontinent stools every 20 to 30 minutes, or by the silent decline of hemoglobin or hematocrit to a level lower than that expected from simple hemodilution. In nine patients there was prolonged violent hemorrhage indicated by copious continued hematemeses or melena, or persisting hypotension despite transfusion of large quantities of blood. In seven of these there had been multiple preceeding hemorrhages.

The most impressive feature of the hospital courses of these patients, was the multiple recurrent bleeding episodes. The number of recurrent hemorrhages ranged from none to ten, with a median of three (Table 2). Many of the 12 patients who died with one or no recurrent hemorrhages were abandoned because of old age or died from other causes such as myocardial infarction before they had time to bleed again. Only two patients had single violent prolonged hemorrhages which provided ample warning of the serious nature of the illness. It seems then that death results from cardiovascular embarrassment following repeated blood losses of from 1,000 to 2,000 cc. which have been replaced inadequately. That the passing of some time is necessary for deterioration to take place, is indicated by the fact that the median day of death was the 6th after admission.

The classic case of fatal bleeding ulcer then, will have four or five short-lived, vigorous, self-limiting

hemorrhages. One occurs during the week before hospitalization. One results in hospitalization. Three will occur at 12 to 48 hour intervals in the hospital. By the time the bleeding is recognized, it will have stopped in 80 percent of the episodes. In those in whom it continues, whether slowly or violently, there will be little difficulty in subsequent recognition.

As reported by others, about three fourths of the cases were in males. Below age 65, the male: female ratio was 19 to one.

Only five deaths occurred in patients under 55 years of age, and three of these followed technical procedures now discarded by most surgeons. Death should rarely occur in men under 55 or in women under age 65—a decade later than suggested by most authors.

TABLE 4. NATURE AND SITE OF LESIONS ACCORDING TO SEX

	Male	Female
Acute gastric and gastritis	1	2
Chronic gastric	9	6
Chronic duodenal	24	4
Stomal	2	0
Unknown (1 after autopsy)	2	0

Associated Diseases. In only 15 patients was there no significant associated diseases diagnosed; four were over age 70, and nine were over age 60. Others have observed the high incidence of associated diseases. Mitty *et al.* showed how poorly patients with general medical disorders tolerate gastrointestinal bleeding. Inability to compensate for repeated blood losses accounts for the high mortality after age 55, rather than any difference in the ulcer or severity or frequency of recurrent hemorrhage.

Nature and Site of Lesions. The incidence of chronic duodenal ulcers in men (Table 4) corresponds with that observed by Coghill and Willcox. As in previous reports, acute gastric ulcers rarely caused fatal bleeding. The history, of course, did not always indicate chronicity of the ulcer. Eight patients subsequently shown to have chronic ulcers had short or inconclusive histories of dyspepsia.

Assessment of Management

Since these patients had died, the treatment had not achieved its purpose. It seemed worthwhile therefore, to critically analyze these experiences with all the advantages of hindsight. In this way difficulties in management were identified for special consideration in the consequent plan of therapy.

By reviewing all details relevant to the number and nature of recurrent hemorrhages, speed and volume of blood given, response of the patients' pulse rate, blood pressure and urine outputs, response of hemoglobin levels to transfusions given and evidence for or against further bleeding episodes, the adequacy of volume and rate of transfusion were evaluated.

In 44 patients, the volume of blood given was considered inadequate. The amount of blood required on admission was regularly underestimated by nearly one half. Commonly, hemoglobin levels of six to eight grams and hypovolemia that these levels imply (but by no means exactly reflect), were accepted. Patients were not provided with a reserve of blood for subsequent recurrent hemorrhages. This failure to provide the volume of blood necessary was thought to be the most important factor in the unsuccessful management of the patients.

The rate of transfusion was frequently found to be inadequate. In only nine instances, excluding operating room transfusions, was blood given rapidly (1,000 cc./hour), and most received rapid transfusions only in immediately preoperative resuscitation. There was regularly a delay in instituting transfusion. This was worst on the patient's arrival at the hospital, when 2 to 4 hours frequently elapsed before blood was actually given.

In assessing the need for operation it was assumed that two recurrent (in-hospital) hemorrhages or violent continued hemorrhage demanded emergency surgical intervention; otherwise it was believed that there was no reason for an emergency (though not necessarily an elective) procedure. Failure to perform indicated operation, or poor timing of operation occurred in 58 percent of the patients. In 21 instances, operation was thought by the reviewers

to have been indicated clearly, but was not carried out. In eight patients it was indicated considerably earlier than it was carried out. The responsibility for the failure to perform indicated surgery was by no means invariably due to the medical attendant. In 19 of 29 patients it was the surgeon who failed to recognize his responsibility to operate at the necessary time. In four instances, there was doubt whether operation was justified at the early stage at which it was carried out.

In 13 patients death was a result of technical surgical complications. Five bled from retained ulcers. Nine suffered fistulas or injury to abdominal organs (e.g., acute pancreatitis). In four patients, operation was performed impulsively with no reasonable attempt to correct blood volume, water or electrolyte deficits.

Table 5 describes the number of physicians involved in the management of these patients. Experience gained from these fatal cases was so widely distributed over the 9 years and 85 physicians that there was little hope that lessons from one patient would be remembered when the next patient was encountered. Also, all too often the responsibility of assessing the patient's condition or blood requirements was left to the woefully inadequate experience and comprehension of a nurse or junior intern.

TABLE 5. PHYSICIANS INVOLVED

<i>Physicians</i>	<i>Residents</i>	<i>No. Patients Seen</i>
54	30	1
19	4	2
6	3	3
5	2	4
1	0	6
—	—	—
Total 85	39	50

Plan of Therapy

The following plan of therapy is based on these observations of the natural history of fatal bleeding ulcer. Provided he is adequately transfused, the patient will survive the admission hemorrhage and the first recurrence in the hospital. There is then no indication for immediate operation simply because of the hemorrhage at admission. Such immediate operation is unnecessary (as a life-saving procedure) since 75 percent of patients will have no further bleeding. The remaining 25 percent can be expected to survive the first recurrent hemorrhage.

Thus the initial treatment regardless of the age or sex of the patient, or the nature of the lesion, is simply adequate transfusion. Since bleeding will usually have stopped by the time the physician examines the patient, the only concern is adequate estimate of blood loss and then, if necessary with central venous pressure monitoring, aggressive replacement. Rapid transfusion is as vital to allow the patient to withstand the loss of 1,500 cc. of blood in the next hemorrhage, if it occurs, as to assist recovery from the previous one.

If another significant hemorrhage occurs in the hospital, it must be feared that the patient will have further recurrent hemorrhages for which he will not be able to compensate. If he is older than 55 and has a chronic ulcer, emergency operation is indicated, not as a rule to stop the bleeding but to prevent further bleeding episodes in the current series. If there are associated general diseases that make the patient less tolerant of blood volume reduction, then operation is even more necessary. It is essential that a 1- or 2-hour period of intense resuscitation precede operation.

Since fatal hemorrhage seldom resulted from an acute ulcer or in patients under 55 years of age, such patients may be transfused through the first hospital recurrent hemorrhage, and operation delayed until after a second recurrence. In this way some unnecessary operations may be avoided. Similarly, the rarity of fatal acute gastric ulcers challenges the role of "blind" gastrectomy.

The number of deaths resulting from operative injury, fistulas and retained ulcers indicates the need to restrict this operative surgery to those who are experienced. Suture-ligation, vagotomy and pyloroplasty seem to be indicated when the operation appears technically difficult, although recent evidence suggests that a high rate of recurrence follows this

procedure in the very patients who have complicated lesions.

The final feature of management should be a "hematemesis team" which would treat patients with gastrointestinal bleeding in a hospital. It has been seen how the lessons, the errors and the understanding were forgotten between cases. The enthusiasm of such a team should alone improve results. It is believed that the concentrated experience of a team, together with its greater interest, might have saved the lives of many of these patients.

Summary

Fifty consecutive deaths from bleeding peptic ulcer, occurring during a 9-year period, have been reviewed.

The natural history of fatal bleeding ulcer was one of multiple, recurrent, abrupt, self-limited hemorrhages. The median number of hemorrhages before death was five, and the median day of death was the sixth after admission. Death seldom occurred in patients under 55 years or in the absence of a chronic ulcer. Death appeared to result from inability to compensate for repeated, inadequately-corrected blood losses.

Review of management showed the most common errors to be: inadequate transfusion, both in volume and rate (88%), failure to operate or improper timing (58%) and avoidable technical surgical complications (26%). The attending physician's infrequent exposure to patients with fatal bleeding ulcers was also an important factor.

A method of management characterized by aggressive transfusion and selective operation is described; most important is a "hematemesis team."

(Table 3 and the references may be seen in the original article.)

THE SIGNIFICANCE OF CARDIAC MURMURS IN ANEMIA

A. A. Dawson MB (Aberd.) MRCPE, Lecturer and K. N. V. Palmer MA MD (Camb.) FRCP, Senior Lecturer, Department of Medicine, University of Aberdeen, Aberdeen, Scotland. Amer J Med Sci 252(5):554-557, November, 1966.

Laennec, in 1846, and Hope, in 1839, both mentioned inconstant cardiac murmurs in chlorosis, and, although cardiac murmurs are frequently heard in anemic subjects, there is often uncertainty whether or not the murmurs are due to anemia. We have

studied anemic subjects clinically, before and after their hemoglobin levels have been raised, to determine the incidence and characteristics of cardiac murmurs in anemia. This clinical approach was adopted, because Weaver and Walker found stetho-

scopic analysis to coincide almost exactly with phonocardiographic findings, with respect to intensity, timing and frequency of murmurs.

Subjects and Methods. There were 72 patients, all with subacute or chronic anemia, most commonly dyshemopoietic or due to malignant reticuloendothelial disease. No patient with recent acute blood loss was included.

The hemoglobin content of capillary blood was measured by the cyanmethemoglobin method, using a single "E.E.L." automatic hemoglobinometer, calibrated so that 14.6 gm. per 100 ml. blood was equivalent to 100% hemoglobin. Blood samples were obtained by needle-stab of a finger by technicians experienced in collecting capillary blood. In all patients, the hemoglobin was less than 65% (9.5 gm. per 100 ml.), the mean hemoglobin being 48.3% (S.D. 10.6).

Auscultation of the heart was carried out, first with the patient recumbent, then sitting up, and during the phases of respiration. Systolic murmurs were classified as midsystolic ejection murmurs, and pansystolic regurgitant ones (Leatham), and, by intensity, into four grades. The subjects were re-examined in the same manner after there had been a rise of hemoglobin of at least 30%, either as a result of therapy with hematinics, or by blood transfusion. In the latter case, re-examination was carried out not less than 2 days after transfusion. The mean hemoglobin level, after therapy, of those patients who initially had cardiac murmurs, was 80.1% (S.D. 11.8).

Results. Forty-six subjects (63.9%) had one or more murmurs. The mean hemoglobin of the subjects with murmurs was 45.8% (S.D. 11.1), and of those without, 52.9% (S.D. 7.9). This difference is significant ($0.02 > p > 0.01$). The mean age of the group with murmurs was 57.9 years (range 22 to 83), and of the group without, 54.1 years (range 24 to 79); this difference is not significant ($0.4 > p > 0.3$).

TABLE 1.—INCIDENCE OF MURMURS RELATED TO HEMOGLOBIN LEVEL

Hemoglobin (%)	Number of Subjects	Murmur (s) Present
15-24	2	2 (100%)
25-34	8	7 (87.5%)
35-44	12	8 (66.7%)
45-54	30	20 (66.7%)
55-64	20	11 (55.0%)

From Table 1, it can be seen that the incidence of murmurs rose as the hemoglobin level fell. However, of the other factors which might cause murmurs, only cardiomegaly and myocardial ischemia were much commoner in those with murmurs than in those without (Table 2).

TABLE 2.—FACTORS OTHER THAN HEMOGLOBIN LEVEL IN INCIDENCE OF MURMURS

	Patients with cardiac murmurs (46)	Patients without cardiac murmurs (25)
Fever (over 99°)	15 (32.6%)	5 (20.0%)
Cardiac failure	8 (17.4%)	7 (28.0%)
Myocardial ischemia	19 (41.3%)	7 (28.0%)
Cardiomegaly	27 (58.7%)	5 (20.0%)
Hypertension	4 (8.7%)	4 (16.0%)
Overt atherosclerosis	8 (17.4%)	2 (8.0%)
Rheumatic heart disease	1 (4.4%)	0

Before correction of anemia, there were 58 murmurs, and 12 patients had two or more murmurs. As a result of raising the hemoglobin, the murmurs disappeared in 18 patients, became less (in number or intensity) in 10, and remained unchanged in 18. We define a hemic murmur as one which is present in the anemic state and disappears when the anemia has been treated. Those with hemic murmurs were, on average, 20 years younger than the other subjects (Table 3).

CHARACTERISTICS OF MURMURS. (1) Forty-one (70.7%) were midsystolic ejection murmurs [23 (56.1%) Grade 1, 12 (29.3%) Grade 2, and 6 (14.3%) Grade 3]. The position of maximal intensity was at the base in 19 (46.3%) and at the apex in 13 (31.7%). Fifteen (36.6%) were reduced by sitting up and 19 (46.3%) diminished on deep inspiration.

(2) Fourteen (24.1%) were pansystolic regurgitant murmurs [4 (28.6%) Grade 1, 5 (35.7%) Grade 2 and 6 (42.9%) Grade 3]. They were almost all best heard at the cardiac apex (92.9%). They were much less affected by posture and respiration, three, only, being reduced by sitting up and one by deep inspiration.

(3) There were 3 early diastolic murmurs, all Grade 1. They were all best heard at the left sternal edge, were all increased by sitting up and diminished by deep inspiration.

TABLE 3.—FATE OF MURMURS IN RELATION TO HEMOGLOBIN LEVEL BEFORE AND AFTER TREATMENT

Fate of Murmurs	Number of Patients	Mean Age (years)	Mean Hb (%)	
			Before treatment	After treatment
Disappeared	18	45.5	47.1	77.0
Got less	10	65.7	40.3	74.7
Unchanged	18	65.9	47.1	84.7

When the anemia had been treated 27 (46.6%) murmurs disappeared. Eighteen were ejection midsystolic, mainly Grade 1 and equally basal and apical, 7 were apical pansystolic regurgitant murmurs of Grade 2 and 3 intensity and two were early diastolic murmurs (the diastolic murmur which persisted was in a patient with rheumatic heart disease). Almost half (44.4%) of the hemic murmurs were reduced by sitting up and on deep inspiration, whereas of the persistent murmurs fewer were diminished by sitting up (22.6%) and on deep inspiration (19.4%).

Discussion. In this study, murmurs occurred in about two-thirds of anemic subjects, the incidence increasing as the hemoglobin level fell, but only a quarter of the subjects had hemic murmurs. Although the presence of murmurs in general was not related to age, those with hemic murmurs were 20 years younger than those whose murmurs were due to other causes.

The hemic murmurs were predominantly soft, ejection midsystolic murmurs, heard equally at the base or apex. Less frequently, they were louder pansystolic regurgitant ones, heard at the apex. Hemic diastolic murmurs were found in two severely anemic elderly subjects. Hemic murmurs became less on sitting up and on inspiration much more frequently than did nonhemic ones.

Diastolic murmurs in severe anemia, without pathological evidence of valvular disease, have been known to occur since 1861 (Friedreich), and were relatively common in the days before treatment of pernicious anemia; for instance, Goldstein and Boas, in 1927, found that 6 of 39 patients with pernicious anemia had diastolic murmurs.

In subjects with hemic murmurs in our series, half had cardiomegaly, without other clinical evidence of cardiovascular disease, whereas in patients with nonhemic murmurs, cardiomegaly was more frequently associated with cardiac failure, hypertension and clinical evidence of atherosclerotic heart disease (Table 4).

TABLE 4.—CHANGES IN MURMURS IN RELATION TO EXISTING DISEASE

	Patients whose murmurs were	
	disappeared (18)	less or unchanged (28)
Fever	6 (33.3%)	9 (32.1%)
Cardiac failure	2 (11.1%)	6 (21.4%)
Myocardial ischemia	5 (27.8%)	14 (50.0%)
Cardiomegaly	9 (50.0%)	18 (64.3%)
Hypertension	0	4 (14.3%)
Evidence of overt atherosclerosis	2 (11.1%)	6 (21.4%)
Rheumatic heart disease	0	1 (3.6%)

It has often been assumed that, as both cardiomegaly and murmurs are common in severe anemia, the murmurs are caused by cardiac enlargement, but Hunter showed that the association between the two was inconstant.

Cardiac enlargement in anemia seems to be due to a combination of cardiac hypertrophy and dilatation (Cabot and Richardson), and, perhaps, fatty changes (Hunter). Although it is not closely related to the severity of the anemia, Sanghvi, Misra and Banerjee concluded that it is very likely to be present if the hemoglobin is less than 8 gm. per 100 ml. blood, and that, in three-quarters of patients, it regresses while the hemoglobin is still significantly below normal.

Murmurs, on the other hand, are more persistent, and may disappear only after the hemoglobin level has been normal for several months (Hunter). This is in accordance with the finding of Tandon and Katiyar that ballistocardiographic abnormalities are present even after improvement in hemoglobin. Whilst cardiac enlargement is commonly found in patients with hemic murmurs, other factors, such as

reduced blood viscosity (Garb) and high cardiac output (Sharpey-Schafer) probably also contribute to the production of these murmurs.

Summary. Seventy-two patients with hemoglobin levels of less than 9.5 gm. per 100 ml. were examined for cardiac murmurs. Murmurs occurred in two-thirds of subjects, but only 46.6% of these mur-

murs were hemic, that is, disappearing as the hemoglobin level rose. The subjects with hemic murmurs were younger, and had cardiomegaly more often unassociated with organic cardiovascular disease than in the anemic subjects with nonhemic murmurs.

(The references may be seen in the original article.)

MEDICAL ABSTRACTS

MILITARY RETIREMENT: THE RETIREMENT SYNDROME

MAJ J. S. McNeil BSc USAF and COL M. B. Giffen MC USAF, (From the Department of Psychiatry, Wilford Hall, USAF Hospital, Aerospace Medical Division, Lackland Air Force Base, Texas.) *Amer J Psychiat* 123:849-854, January 1967.

Considering that the typical military officer retires from the military service at age 45 and enlisted men usually at a somewhat earlier age, as noted in this article, military retirees do not conform to the Symbolic Meaning of Retirement. The latter is perceived by the authors as a withdrawal or disengagement from active full-time paid employment at age 65 or older by an individual who has made his major contribution to society and whose occupational pursuits, if engaged in, are to be avocational.

The majority of military retirees at the time of retirement have a wife and two or three dependent school-age children and while the military retirement pension is generally considered to be liberal, it is not sufficient in most instances to provide adequately for the retiree and his family at their accustomed standard of living. This then, in addition to the demands of society that able-bodied middle-aged men be constructively employed and the fact that most men feel an emotional need to work, make career military personnel accept the idea of seeking employment as the normal procedure. After this beginning, the authors discuss military service culture as it differs from nonmilitary society and its influence on the thinking of the retiree when he is faced with a radical change from the way of life he has known. This may be accompanied by anxiety or depression which may precipitate certain symptoms which are referred to as the Retirement Syndrome. Listed among these are: irritability, loss of interest, lack of energy, and reduced efficiency. Increase in alcoholic intake may occur. Psychoso-

matic symptoms usually centered around the gastrointestinal tract or the cardiovascular system may develop. If psychoses are in evidence, the diagnoses are generally in the class of depressive reactions or the paranoid group. Aggravation of preexisting medical symptoms may recur.

A fairly distinct transitional period following retirement is described which in previous publications, these same authors have labeled a time of role confusion. This period is marked by feelings of anxiety and uncertainty associated with loss of identity, for often relationships are dramatically different from those experienced in military service and there may be loss of status. Happily, the transitional phase may be negotiated successfully and satisfactory adjustment to retirement made but if the struggle to readjustment is too great, maladjustment occurs and symptoms and/or problems requiring professional intervention result.

Illustrative case histories are presented. The authors urge early identification of retirement-related problems so that proper treatment can be initiated before the psychopathology has acquired a flavor of permanency.

PULMONARY HISTOPLASMOSIS IN A FARM FAMILY—FIFTEEN YEARS LATER

J. J. Procknow MD, (From the Department of Medicine, University of Chicago.) *Amer Rev Res Dis* 95:171-188, February 1967.

This interesting and carefully done study of a farm family six members of which were directly exposed to dust infected with *Histoplasma capsulatum* and acquired pulmonary histoplasmosis and two members which were not directly exposed and did not develop the disease is well worth reading. The present report spans more than 15 years of observation of this group. All members of the family are still living and are well clinically.

The infections have been attributed to the inhala-

tion of histoplasma—laden dust wafted into the air during the cleaning of an unused silo. *Histoplasma capsulatum* was cultured from the silo soil in 1950 (historically, this proved to be the first isolation of the fungus from a point source responsible for an epidemic in which the etiologic agent was also isolated from an acutely infected person). Forty-four separate collections of soil have been made during the past 15 years and of 1,561 individual soil samples, 136 (8.6 percent) were positive for *Histoplasma capsulatum*.

The clinical spectrum of the mycotic infection ranged from asymptomatic hilar adenopathy to life threatening extensive pulmonary involvement with dissemination. The father, who experienced the most severe pulmonary infection, still has symptoms of exertional dyspnea and excessive sweating. Remarkable healing of the pulmonary lesions and lymph nodes by fibrosis and calcification has occurred.

The authors purpose in this report is: (1) to define the present clinical and roentgenographic status of members of the farm family and (2) to emphasize the restricted localization and persistence of *Histoplasma capsulatum* as a saprophyte in soil as determined from cultural surveillance of the silo soil for the past 15 years.

POSSIBILITY OF A NEW INFECTIVE AETIOLOGICAL AGENT IN CONGESTIVE CARDIOMYOPATHY

M. V. Braimbridge FRCS, Sally Darracott FRMS,
J. Chayen D Sc, Lucille Bitensky MC Path, L. W.
Poulter FRMS. *Lancet* I: 171-176, January 28,
1967.

Clinically cardiomyopathy presents, according to the authors, in an obstructive form with gross hypertrophy of the out-flow tract of the left ventricle, in a constrictive form due usually to myocardial infiltration by generalized disease processes, and in a congestive form in which cardiac dilatation and failure are the dominant features and a fluctuating down hill course the rule. Investigation of the congestive form is by exclusion of ischemic, rheumatic, and generalized disease and by an attempt to incriminate a specific factor which commonly is negative. They refer to examination by others of specimens of human left ventricular myocardium by drill biopsy at the time of cardiac operations which demonstrated biochemical and biophysical changes which were specific to cardiac failure and which sug-

gested the addition of specific coenzymes in its treatment. The possibility of making an accurate diagnosis and instituting some specific treatment in a lethal disease, in their opinion made left ventricular biopsy in the investigation of cardiomyopathy ethically justifiable.

The following is the authors' summary:

"Specimens of myocardial tissue, taken from the apex of the left ventricle at biopsy and/or at necropsy from seven patients with congestive cardiomyopathy, have been examined by a variety of histological and biological techniques. Sections of myocardial tissue taken from all seven patients contained peculiar structures; and these structures were seen in only one out of a hundred and twenty specimens taken from patients undergoing surgery for other lesions. The structures were recognized by their metachromatic staining, and were pleomorphic. The larger structures, termed "mark bodies", frequently appeared as a halo around swollen nuclei. Biochemical tests indicated that the structures were living but foreign to the myocardial tissue. The agent could not be grown by conventional bacteriological procedures, but did grow when specimens of tissue were placed in maintenance culture, with or without certain additives, and at high oxygen tensions. Injections of cardiomyopathic tissue proved fatal to mice and metachromatic particles could subsequently be identified in these animals. Thus Koch's four postulates are satisfied and it seems that there may be an aetiological infective agent in human congestive cardiomyopathy."

In their conclusion they say that if satisfying Koch's postulates is accepted, it is possible that human cardiomyopathy may commonly be associated with an infective agent and their object is to draw attention to this possibility and to emphasize that special conditions, both histological and cultural, may be required to demonstrate whether such an infective agent is indeed concerned in the etiology of congestive myopathy.

EMPHYEMA AND SURVIVAL FOLLOWING SURGERY FOR BRONCHOGENIC CARCINOMA

B. Cady MD and E. E. Clifton MD, (From the Department of Surgery, Thoracic Service, Memorial Hospital for Cancer and Allied Diseases and the James Ewing Hospital [of the City of New York].) *J Thorac Cardiovasc Surg* 53: 102-108, January 1967.

Sporadic reports of the oncolytic effect of viral and bacterial organisms and products made for years

and recent speculation in the literature concerning the increased survival of patients with empyema following resections for bronchogenic carcinoma prompted the authors to review such cases at Memorial Hospital. Careful analysis of their series of postoperative empyemas complicating resections for bronchogenic carcinoma failed to reveal an increase in five year survival. However, bacteriological study of patients who did survive five years revealed that they tended to have more streptococcal and staphylococcal infections and with larger numbers of organisms. They conclude that with clear-cut laboratory evidence of potent oncolytic activity by virus and bacterial products, clinical application needs to be continually reassessed.

IATROGENIC PULMONARY EDEMA IN SURGICAL PATIENTS

J. Adriani MD, R. Zepernick MD, W. Harmon MD, B. Hiern MD, (From the Department of Surgery, Tulane University School of Medicine, The Department of Surgery, Louisiana State University School of Medicine, and the Department of Anesthesia, Charity Hospital [New Orleans, Louisiana].) Surgery 61:183-191, February 1967.

The authors admit to deliberate oversimplification and to being dogmatic in their explanations of underlying factors causing lung edema in order to emphasize the iatrogenic features of the syndrome and to focus attention upon a particular attribute of an offending agent. They list precipitating factors of pulmonary edema compiled from the literature as follows: "(1) incomplete cardiac emptying; (2) shift of blood from the periphery to the pulmonary vascular bed; (3) circulatory overload, which results from an increase in blood volume beyond normal limits by overtransfusion or infusion of fluids; (4) negative pressure on the airway, which increases the gradient between the transmural capillary pressure and the alveolar pressure and favors transudation; (5) idiopathic—situations in which no incriminating factor is apparent come under this category; and (6) injury to the alveolar membrane by noxious substances. An increase in the distance between the alveolar and capillary walls is invariably present in pulmonary edema. Varying degrees of alveolar capillary block result, and this interferes with gaseous exchange. The resulting hypoxia initiates a vicious cycle which is difficult to disrupt and which aggravates the condition."

In their discussion they stress the fact that pulmonary edema is seldomly caused by one single factor, rather a single factor is the precipitating factor of several co-existing factors in pulmonary edema of iatrogenic origin as well as in edema caused by disease. Iatrogenic pulmonary edema in surgical patients, they have found, has been precipitated by: "(1) incomplete cardiac emptying due to drugs which depress the myocardium (thiopental, local anesthetics, barbiturate, narcotics), drugs which caused tachycardia (atropine, epinephrine), and drugs which cause persistent severe arrhythmias (cyclopropane, halothane); (2) redistribution of blood from the periphery (vasoconstrictors, central stimulation); (3) negative pressure on the airway (obstruction, bronchospasm); (4) circulatory overload (blood, fluids, absorption of irrigating fluids); and (5) injury to alveolar membranes by noxious inhalants, excretion of poisons (kerosene), and aspiration of gastric contents in attempting to evacuate the stomach in cases of poisoning".

The most frequent causes of iatrogenic pulmonary edema in surgical patients, they say, are: circulatory overload and the use of anesthetics which depress the myocardium, particularly when used in combination with potent vasoconstrictors.

GASTRIC SUMP DRAINAGE WITH A WATER SEAL MONITOR

R. K. Hughes MD, D. G. Wooton MD, (From the Departments of Thoracic and General Surgery, Wadsworth Hospital Veterans Administration Center, Los Angeles and the Department of Surgery, School of Medicine, University of California at Los Angeles.) Surgery 61:192-195, February 1967.

The use of a gastric sump tube with a water seal drainage monitor is described in this article. By comparative studies with gastric drainage through a Levin tube (Wangensteen), the authors found that their double lumen tube has the advantages of easy tube patency determination and improved maintenance of patency.

(The same issue of Surgery 196-202 includes descriptions of "A New Underwater Suction Drainage Unit" by H. J. Engelsher MD, Yonkers, New York, and "An Efficient Low Negative Pressure Suction Drainage Unit" by R. Freake and F. C. Caldwell, Melbourne, Australia.—Editor.)

DENTAL SECTION

A NEW FIXED TEMPORARY DENTAL SPLINT

*CDR W. N. Johnson DC USN and LT J. E. Groat
DC USN, J Western Soc Periodont 14:153-156,
December 1966.*

A new type of fixed temporary splint is presented and detailed instruction on its fabrication is developed. The Johnson-Groat splint utilizes a strip of brass mesh (size 80) as the matrix into which a cold-cure resin is deposited. This type of fixed temporary splint presents a clean, esthetically acceptable, inexpensive and satisfactory immobilization of segments of the inexpensive and satisfactory immobilization of segments of the dental arch requiring temporary splinting.

The procedure for fabrication utilizes the use of the rubber dam to permit clear access to the segment to be immobilized. A strip of the brass mesh is first carefully adapted about the teeth to be immobilized, carefully guiding the mesh into the interproximal spaces with a plastic instrument and then held in this position with interdental ties of either dental floss or very light ligature wire. The mesh should cross the facial surface at the height of contour and the lingual surface at the level corresponding to the buccal, to prevent dislodgment in a gingival or occlusal direction. With the mesh in place, cold-cure acrylic is deposited into the mesh in an incremental fashion until all the mesh is completely covered. Care should be taken not to allow the acrylic to flow into the gingival embrasures, so as to occlude this space and prevent proper home care. The hardened acrylic is contoured and polished prior to the removal of the

rubber dam. After the dam is removed the occlusion is checked and corrected, eliminating all interferences.

Several modifications are presented including a method for bridging edentulous areas. If further information is required, the author's address is U.S. Naval Dental Clinic, Marine Corps Base, Camp Pendleton, California 92055.

(Abstracted by: CAPT P. F. Fedi DC USN.)

SURGICAL MANAGEMENT OF ANKYLOSIS OF THE TEMPOROMANDIBULAR JOINT: REPORT OF TWO CASES

J. S. Lindsay, C. L. Fulcher, H. J. Sazima, and H. G. Green, J Oral Surg 24(3): 264-270, May 1966.

A discussion is presented of the nature and etiology of true (I) and false (II) ankylosis of the temporomandibular joint (TMJ). In I, usually caused by trauma or infection, there is a fibrous or bony union of the articulating surfaces. In II, wound scars or cicatricial bands tend to bind the mandible to various parts of the cranium.

Correct diagnosis demands the careful evaluation of the anamnesis and clinical, radiographic and laminagraphic findings. Two instances of I and II are presented in which treatment consisted of osteotomies in the mandible to create pseudarthrosis at varying distances from the TMJ. Grafts of freeze-dried fascia lata were secured within the osteotomy sites to prevent subsequent bony union.

(From: Oral Res Abs 1(7):649. Abstracted by: Dan J. Kaznelson)

PERSONNEL AND PROFESSIONAL NOTES

NAVY OPENS NEW DENTAL RESEARCH INSTITUTE AT GREAT LAKES

A division of the Naval Administrative Command dental department at Great Lakes was formally activated on February 3 as the Naval Dental Research Institute. The research unit is an independent activity with its own officer in charge, who will report

directly to the Bureau of Medicine and Surgery.

An outgrowth of a dental research project assigned to Great Lakes shortly after World War II, the unit will take on the mission of research, test, evaluation and development in dental and allied sciences. CAPT Gordon H. Rovelstad DC USN, who has spearheaded the Administrative Command's dental research activities since July 1965 has as-

sumed duties as the new institute's first officer in charge.

The activation ceremony, held at 1000 on 3 February 1967 included the opening of a newly designed dental clinic. The unique clinic will enable the institute to study new concepts in equipment, practice and clinic management, particularly as it relates to military dentistry.

RADM Howard A. Yeager, Commandant of the Ninth Naval District, welcomed the unit and introduced the principal speaker of the ceremony, RADM Frank M. Kyes, Assistant Chief of the Bureau of Medicine and Surgery and Chief of the Dental Division. Admiral Kyes spoke on the role that Great Lakes has played in the dental research in the Navy.

In 1947, the Navy initiated a long range dental research project at Bethesda, Maryland, and a year later shifted the operation to Great Lakes.

In later years the Great Lakes investigators focused attention to the specific study of dental caries.

The major effort in this study was aimed at discovering why certain recruits had no dental caries or previous dental or oral diseases. These recruits numbered only one out of every 600 men to enter training at Great Lakes. As a result of these and other studies, new caries can be predicted up to four months ahead of time. The researchers hope to find additional means of preventing dental caries, in addition to the stannous fluoride treatments now being used throughout the Navy.

CAPT Gordon H. Rovelstad DC USN, a native of Elgin, Illinois, received his education in DDS, MSD, PhD, from Northwestern University where he was

assistant professor, Pedodontics and Preventive Dentistry. He has an extensive background in dental research which includes growth and development, caries prevention and control, salivary enzymes and oral enzymes, and cytology of the oral cavity. He has authored many articles, and is internationally known as a lecturer. Among his many contributions he served as Chairman, American Board of Pedodontics and is presently Secretary-Treasurer of the International Association for Dental Research.

PROCUREMENT OF RADIOGRAPHIC FILM

JOINT FMSO-FLDBRBUMED INSTRUCTION 6700.1A CHANGE TRANSMITTAL No. 63 contains information relevant to the authorization of activities to procure locally the various manufacturers' commercially available X-ray film. This establishes a means by which the individual radiologist is able to select the exact type and brand of X-ray film required to meet his professional needs.

REPORTING DEFECTIVE OR UNSATISFACTORY MEDICAL AND DENTAL MATERIAL

Attention is invited to JOINT FMSO-FLDBRBUMED INSTRUCTION 6700.16B, which delineates the procedure for reporting defective or unsatisfactory medical and dental material. Activities are reminded that such reports must be completely objective and not merely based upon personal like or dislike of a particular item.

NURSE CORPS SECTION

NURSE CORPS OFFICERS ATTEND NEUROPSYCHIATRIC NURSING COURSE

A conference on psychiatric nursing was conducted at the Naval Medical School, National Naval Medical Center, Bethesda, Maryland the week of 16 through 20 January 1967. Twenty Nurse Corps officers attended the course, which was presented and directed by CDR Mary F. Cannon NC USNR, LCDR Katherine Wilson NC USN, and LCDR Angeline G. Liakos NC USN.

The conference opened with a consideration of characteristic problems of psychiatric nursing in the Navy. Special problems of mental illness and psy-

chiatric nursing of the pediatric military age group and geriatric patients were presented. The role of the chaplain, psychiatric social worker, occupational therapist, psychologist and psychiatrist in the care of the mentally ill was presented by specialists in these respective fields.

Some time was devoted to consideration of the neuropsychiatric technician course and the assignment of hospital corpsmen as psychiatric aides. Included in the program were lectures which emphasized the function of the nurse in teaching psychiatry and mental health. A resume of the historical evolution of the management of the psychiatric patient was given in a presentation on research in psychiatry.

NURSES' AIDE TRAINEESHIP PROGRAM CONDUCTED AT THE USNH, OAKLAND

During the summer of 1966 the Welfare Department of Contra Costa County, California requested facilities and personnel of Oakland Naval Hospital to train selected welfare recipients as nurses' aides. After careful screening ten women were selected to participate in this program and were provided with uniforms and the necessary equipment to function in a hospital setting.

Formal classes were started on 5 July 1966. These women were receiving welfare in the form of aid to dependent children for families which ranged in size from one child to twelve children. All selected trainees were high school graduates and some listed a year or two of junior college. The trainees were

mature, highly motivated women who managed to attend all classes in spite of overwhelming home problems. The course consisted of 70 hours of formal class room instruction, ward experience, and participation in inservice programs instituted for them, or attendance at the organized inservice program for hospital corpsmen. Frequent counseling sessions were held to evaluate and resolve problems encountered on the wards. At the end of six months this program is considered to be very successful. The Welfare Department has requested Oakland Naval Hospital to continue this program.

Further information concerning classes given for this program may be obtained from Chief of Nursing Service at the Oakland Naval Hospital.



LCDR Ruth Pampush NC USN Instructing Nurses' Aid Trainees at the U. S. Naval Hospital, Oakland, California.—Official U. S. Navy Photograph.

PREVENTIVE MEDICINE SECTION

CASE OF HUMAN BABESIOSIS

USDHEW PHS Comm Dis Cen, Morb & Mort Wkly Rpt 16(1): 8, Jan 7, 1967.

The Communicable Disease Center reports a case of babesiosis occurring in a 46-year-old white male resident of San Francisco, in August 1966. Diagnosis was established both serologically and morphologically. The mature parasites in infected RBCs appear in typical clusters of 4 pyriform organisms arranged in a rosette pattern. The parasites were unpigmented and early forms had spherical and ameboid shapes resembling the trophozoite stages of *Plasmodium* species.

Babesiosis (piroplasmosis) is a common disease of domestic and wild animals, especially in tropical areas. It normally results from the bite of infected ticks but mechanical transmission can occur. In the United States the parasite is usually found in horses and dogs. The case noted is the second documentation of human babesiosis in the world. The first case occurred in Yugoslavia in 1956 and terminated in death. Interestingly, both individuals had undergone splenectomies some years prior to their disease.

The importance of this parasite as a human pathogen is minor, but the fact that it can be confused with malaria and is apparently a zoonotic disease presents some points of interest to medical personnel.

TRANSFUSION INDUCED MALARIA NEW YORK CITY

T. Fodor MD, et al, Morb & Mort Wkly Rpt 15(52):451, Dec 31, 1966.

A case of blood transfusion induced malaria in a 64-year-old man has recently been reported from New York City. The patient had onset of chills and fever on 29 October 1966; *Plasmodium falciparum* parasites were found in blood smears. He had not traveled outside the United States since he emigrated from Italy in 1913 and he did not have a history of self-inoculations. Because of continuous massive bleedings from the renal pelvis, the patient had received 70 units of blood over the 2-year period prior to onset of malaria.

During the 2 months preceeding the onset of illness he received 2 units of blood on 6 and 20 September and on 14 October 1966. Five of the 6 blood donors were located and none of them had a history of malaria, overseas travel, blood transfusions or drug addiction. The 6th donor was identified as a 28-year-old male Ghanaian who had resided in New York City from July through November 1966. He had donated blood on 14 October. At the time of the investigation, the donor had returned to Ghana. The blood bank records do not indicate a history of malaria in this donor, but malaria is known to be endemic in Ghana.

Editorial Note: Since 1957, 10 cases of blood transfusion induced malaria have been reported to the Communicable Disease Center. Of these, 7 cases were due to *P. malariae*, one to *P. vivax*, one to a mixed infection of *P. malariae* and *P. falciparum*, and in one case the plasmodium species is unknown. In only one instance was the infectious blood donor identified (New York City, 1958).

SHIGELLOSIS IN THE UNITED STATES

Los Angeles Co. Hlth Dept, Calif, Morb & Mort Wkly Rpt, p. 1, Nov 26, 1966.

The Shigella Surveillance Program of the Communicable Disease Center, Public Health Service, Atlanta, Georgia, began in the fall of 1963, with 13 states reporting shigella isolations. Now, all 50 states are reporting, along with the Virgin Islands, New York City, and the District of Columbia. These data have been analyzed quarterly since the inception of the program, and, through the 2nd quarter of 1966, there have been almost 19,000 reported isolations of shigella.

This reporting system has contributed epidemiological data on shigellosis in the United States. The total number of isolations reported to the Communicable Disease Center to date is 18,983. The average number of isolations reported quarterly since nationwide reporting has been attained is 2,000. The total number of isolations from nonhuman sources is 126.

It is stressed that the reported isolations tabulated do not coincide with the true extent of shigellosis. There are many reasons for this; underreporting of

isolations from shigellosis or some reported isolations which come from asymptomatic carriers.

Analysis of Data

Two-thirds of the isolations have been from children under 10, and almost 40% of the isolations have been from children in the 1-4 age group. Although no overall sex predilection exists, there has been a significant preponderance of isolations from males 1 year of age as differences are significant at P equals less than 0.0001 when the standard error of the percentages is analyzed. These discrepancies may represent unknown bias in the reporting system rather than the disease itself. However, predominance of male infants and children has been reported in other diseases, such as staphylococcal skin infections, purulent meningitis, infectious croup, neonatal sepsis, and ECHO 9 central nervous system infections.

Speculative theories to account for the marked preponderance of isolations from females over 20 have been advanced, the most popular being that women of this age are in closer contact with children than are men of this age. The Salmonella Surveillance Data from the Communicable Disease Center correlates with these findings: that is, there is a marked predominance of isolations from males under 20 years of age, and a marked predominance of isolations from females over 20.

S. sonnei has accounted for 37.1% and *S. flexneri* 2 for 25.4% of the total isolates. Each quarter, a total of 6 strains accounts for over 85% of all isolates. Isolations of *S. flexneri* 5, *S. boydii*, and *S. dysenteriae* have been rare.

The greatest number of shigella isolations reported occurs in the fall. With a delay of several weeks in reporting this indicates a peak incidence of shigellosis in late summer. The isolation of *S. flexneri* shows the greatest seasonal variation. The ratio of *S. flexneri* to *S. sonnei* isolations varies in different parts of the country. *S. flexneri* accounts for $\frac{2}{3}$ of total isolations in the South, but less than half of the total isolations in the North. *S. sonnei* is most commonly isolated in the northwestern United States, whereas in Canada, *S. flexneri* is more commonly isolated; indicating, perhaps, that climate is not the only factor in the "regionalization" of serotypes. Other regional trends exist. For example, 33 of the 58 isolations of *S. dysenteriae* 2, reported since 1963 are from one state in the Great Lakes area.

There were 126 isolation of shigella from non-human sources. Primates account for more than 80% of these isolations. Another interesting source

has been the fluid contents of plastic ice cubes imported from the Far East. There have been a few isolations of shigella from fowl products, including one isolation of *S. sonnei* from frozen egg albumin in a northwestern state and several isolations of shigella from checked and frozen eggs in a western state. No reports have been received of human shigellosis transmitted from any of these nonhuman sources, although human shigellosis has been transmitted from primates.

Discussion

Shigellosis is endemic in certain medical institutions and Indian reservations, but is also present in many lower socioeconomic communities across the nation. A recent investigation of shigellosis in a large northwestern city showed that the majority of cases occurred in lower socioeconomic groups, and that transmission was by person-to-person contact, primarily among children.

In recent years, there has been growing interest in the possible control of shigellosis in confined groups by immunization with a mutant or hybrid, living, attenuated, polyvalent, oral shigella vaccine, such as that being developed by Formal and colleagues at Walter Reed Army Medical Research Center. One such vaccine, prepared from a streptomycin-dependent *S. flexneri* 2a organism, has protected Yugoslavian soldiers against dysentery caused by *S. flexneri* 2a. To better define the scope of the problem in confined groups, the Enteric Diseases Unit of the Communicable Disease Center has emphasized surveillance and investigation of shigellosis in institutions for the mentally retarded. A common pattern in these institutions is that children, especially the more retarded children, have the highest incidence of shigellosis. Newly admitted children are likely to develop shigellosis within the first few months. Despite constant efforts to improve hygiene, shigellosis remains endemic. Therefore, an effective vaccine could greatly facilitate control of institutional shigellosis.

(The references may be seen in the original article.)

FOODBORNE ILLNESS IN THE UNITED STATES

USDHEW Release in P.M. Papers, June 14, 1966.

Most cases of foodborne illness in the United States are preventable but continue to occur because people preparing and serving food fail to apply

known food-protection measures. To warn housewives and restaurant chefs of this danger, a new leaflet, "Hot Tips on Food Protection," has been published by the Public Health Service Division of Environmental Engineering and Food Protection.

Among foodborne illnesses which may result from improper cooking, the leaflet points out, are:

Salmonellosis, a food infection coming from the presence of *Salmonella* organisms which may be found in poultry, eggs, and egg products, even if frozen.

Trichinosis, a foodborne disease transmitted to humans through raw or undercooked pork which may have a parasite, *Trichinella spiralis*, in muscular tissue.

Staphylococcal food poisoning, an illness caused by a toxin made by staphylococcus organisms sometimes present in cream-filled pastries and custards.

Botulism, a sometimes fatal disease, caused by *Clostridium botulinum* organisms (commonly present in soil) which, unless destroyed by proper canning techniques, may form a toxin in certain canned foods.

The leaflet explains how proper cooking and handling of foods which have often been incriminated in outbreaks of foodborne illness may help to eliminate these and other kinds of food poisoning.

Individual free copies may be obtained from the PHS Public Inquiries Branch, Washington, D.C. 20201, or from Regional Offices of the U.S. Department of Health, Education, and Welfare (Att: RPH Regional Program Director, DEEFP) in Boston, New York, Charlottesville (Va.), Atlanta, Kansas City (Mo.), Chicago, Dallas, Denver, and San Francisco.

RICP—AN EXPERIENCE IN VIRGIN ENTOMOLOGY

J Econ Entom 58(5):1152, Dec 1965.

In April 1951, the U.S. Government responded to a cry for help from Iran and helped change a way of life for a major portion of the world's population.

Iran was facing the prospects of the heaviest locust outbreak in 80 years. The assistance supplied was in the form of 8 small spray planes, 13 tons of insecticide, and an entomologist well trained in modern ways to battle insects. These modern methods soon had the locusts under control.

The successful locust program in Iran was closely observed by representatives from India and Pakistan. These countries requested similar assistance from

the United States—starting a chain of events that led to similar agreements with most of the countries of Africa, Asia, and the Near East. These countries had been plagued by locusts for centuries.

Within a few years, this international cooperation had reduced the historic locust plagues to a routine surveillance and control program to eliminate incipient infestations as fast as they appear. Literally millions of people are now being fed from the crops previously sacrificed to the locust.

The success against the locust prompted several countries to request further assistance in combatting a myriad of insect pests competing with villagers for food. This led to the development of the Regional Insect Control Project (RICP).

RICP became a formal program with the signing of an interagency agreement between the Foreign Operations Administration in the State Department and the Agricultural Research Service of the U.S. Department of Agriculture in 1954. The project now is a part of the Agency for International Development (AID) programs to assist underdeveloped countries. The Plant Pest Control Division is the implementing agency for USDA. As such, PPC recruits personnel, acts in an advisory capacity, and provides technical direction of plant protection programs. The objectives are:

1. To maintain facilities and provide needed services for coordinated locust control program.
2. To assist the U.S. AID missions in their program to aid cooperating countries in developing and strengthening plant protection organizations through demonstrations of pest control techniques, and training of nationals.
3. To coordinate insect control activities in the U.S. AID programs where such activities involve cooperation with international organizations such as FAO and desert locust control organizations.

Entomologists assigned to RICP faced monumental problems at the start. Native economic entomologists were scarce in most of the countries requesting assistance. Methods of combatting pests were primitive at best. There was no equipment and the lack of rural development made it impractical to follow the progression of events experienced in the United States.

The insect problem was enough to stagger the imagination. In addition to the common insect pests found in the United States, most of these underdeveloped countries must battle insects we have never had to combat. Some of these pests include the spiny bollworm, *Earias insulana* (Boisduval); the senn pest, *Eurygaster integriceps* Puton; the Ba-

luchistan melon fly, *Myiopardalis pardalina* (Bigot); the Egyptian cottonworm, *Spodoptera litura* (F.); and the Durra stem borer, *Sesamia cretica* Lederer—all extremely serious agricultural pests.

Despite these handicaps, each RICP entomologist knew that he must compress 100 years of know-how and progress into a few short months. Thus, each started with the simplest machines, demonstrations, and training in basic entomology.

Ingenuity and resourcefulness are extremely important in developing organized pest-control programs in these countries. Demonstrations of modern pest-control procedures are often carried out in areas where previous attempts to control pests meant driving them from fields with noisemakers, flails, or black magic. Often, the entomologist must first convince the farmer that insects are actually the cause of poor yields before showing him a simple control procedure.

RICP plays an important role in training technicians and selecting trainees to be sent to the United States or other countries for advanced training. This training is paying off. While economic entomologists are still scarce, many underdeveloped countries now have several. Supporting personnel, spray equipment operators, and pilots also are being trained to assist the entomologists.

RICP planes and personnel stationed in Ethiopia are used to supplement the limited resources of the African countries in conducting intensive aerial reconnaissance to locate incipient infestations of desert

locusts before they reach outbreak proportions and, when necessary, to assist in controlling infestations. Planes from this unit are used also to demonstrate aerial application techniques for a wide variety of agricultural pests. Though stationed in Addis Ababa, 1 or all the planes can be moved to trouble spots upon request. The planes include 5 Cubs, 3 Cessnas, and a recently added C-47.

In addition, RICP joins forces with individual countries and the Desert Locust Control Organization for East Africa in conducting aerial surveys to determine whether buildups are occurring in historic locust breeding grounds in remote areas of Ethiopia, Sudan, and West Africa. Prompt control measures in these primary breeding areas can do much to prevent further outbreaks.

It is significant that from the beginning RICP work has been directed toward establishing the country's own capability to cope with locust invasion and other pest problems. As evidence of the success of this approach, several of these countries can now handle most insect outbreaks with a minimum of guidance. Some underdeveloped countries are starting their own survey programs, establishing reference museums, establishing quarantines to prevent spread of pests, and initiating control programs on their own.

Pest control is now being accepted by the farmers. RICP's present task is to assist these countries in developing stable organizations and programs that will continue to carry on the work begun and to offer a helping hand when needed.

ANNUAL SURVEILLANCE SUMMARY MENINGOCOCCAL INFECTIONS—1966

USDHEW, PHS, Wkly Morb & Mort Rpt 16(2), Jan 14, 1967.

The total of 3,373 meningococcal infections reported to the National Communicable Disease Center, Atlanta, Georgia, represents an increase of 10.6% over the 1965 total of 3,051 cases. Most of this increase occurred from March through June 1966; since July the weekly totals have closely followed those of the previous years.

Of the 1966 total, 331 cases (9.8%) were reported from military installations; 80% of these cases were from California, Louisiana, New Jersey, Kentucky, Texas, South Carolina, and Missouri. In

contrast, only 7% of total cases in 1965 were reported from the military.

The 1966 attack rate for meningococcal infections in the United States was 1.7 per 100,000. This represents a continuation of the mild upward trend in attack rates which has been evident since 1962.

Table 1 presents the analysis by serogroup of 776 strains of *Neisseria meningitidis* which were isolated mainly from blood or cerebrospinal fluid in 1966. As anticipated on the basis of experience in 1964

TABLE I
Strains of *Neisseria meningitidis* Submitted to NCDC—1966

Serogroup	Number	Percent
A	2	0.25
B	548	70.6
C	98	12.6
D	2	0.25
Not typed & "Rough" strains	126	16.3
TOTAL	776	100.0

and 1965, the predominant serogroup was again group B, comprising approximately 70% of the total number of strains.

Sulfonamide sensitivity studies revealed that 80%

of Group C organisms were sensitive to 1 milligram percent or less of sulfadiazine whereas only 50% of Group B organisms were inhibited by this concentration.

KNOW YOUR WORLD

Did You Know?

That a new method has been devised to solve malaria protection problem in nomads of the Middle East where DDT spraying of tents and huts has failed to protect people from mosquitoes?

Ordinary cooking salt, medicated with chloroquine, proved successful in a recent pilot project in Iran. The project involved 1,500 to over 17,000 nomads. The parasite rate in the blood of nomads in this project dropped from 18.7% to 3.4% within the first year, and to 0.11% the next year. There were no new cases of malaria reported in persons more than 2 years of age and no signs of resistance were detected. (1)

That the WHO reports the occurrence of influenza-like illness in cities of the central Asian region of the USSR?

Over 30,000 cases have been observed in Moscow in children and adults; outbreaks are clinically mild. Influenza virus B has been isolated, and a few cases due to virus A₂ have been diagnosed. (2)

That an epidemic of louse-borne typhus, laboratory confirmed, affected 10 persons in Zoyatlán de Juárez, Municipality of Alcozauca, Guerrero State, Mexico, from 2 to 27 November 1966?

The disease was brought under control by dis-

insection with DDT and treatment with chloramphenicol. (3)

That a severe outbreak of gastroenteritis with 5,000 cases and 40 deaths has occurred in Guyana during January 1967?

The etiology of the disease is still unknown, as tests for bacteria have had negative results. There were a few cases of sudden unexpected death without dehydration, a number of cases with convulsions, and some with post-mortem evidence of encephalitis. (4)

That 61 cases of psittacosis in man were reported from 22 U.S.A. states in 1965, 8 cases more than in 1964?

Parakeets were most often incriminated as the source of human infection, with pigeons next. Of 23 human cases, 12 were traced to pet bird owners, and 5 in pet bird breeders or dealers. Two turkey ornithosis outbreaks were reported in 1965: 1 in Minnesota with 3 human infections and the other in Virginia, in 1 human case. (5)

That the 1967 budget for the World Health Organization is \$51,515,000? (6)

That the leading causes of death in France in 1964 were: 19.6%—heart diseases; 18.8%—cancer;

13.3%—kidney disease; 10% violent deaths; 6.1%—respiratory diseases; 5.9%—cerebral lesions; 2.3%—infectious diseases; 1.7%—tuberculosis alone; all other diseases account for 22.3% of deaths? (7)

That the WHO airlifted vaccines for the protection of 50,000 people against paratyphoid and typhoid epidemics to counter the threat of outbreaks in the flood-devastated northern regions in Italy? (8)

That the First Chilean Seminar of Food Technology was held in Santiago de Chile from 20 to 23 October 1966?

This seminar was organized by the Chilean Society of Food Technology, the Institute of Sciences and Food Technology of the University of Chile, and the Food and Agriculture Organization (FAO) USA, of the United Nations. (9)

That European bees are forbidden to enter Australia?

In the interest of the continuing good health of Australian bees, their European counterparts are banned under newly amended quarantine regulations. The Australian Northern Agricultural Region apiary officer said the regulations are designed to prevent the introduction of "Isle of Wight" disease to the island continent. However, bees from Canada, the United States and New Zealand, where the disease is unknown, are still welcome. (10)

References

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7. JAMA 199(3):229/197, Jan 16, 1967 (News Briefs, France, Nov 12, 1966).
8. JAMA 199(3):230/198, Jan 16, 1967.
9. JAMA 199(4):292/140, Jan 23, 1967.
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EDITOR'S SECTION

EIGHTEENTH ANNUAL JOINT SCIENTIFIC MEETING

The Eighteenth Annual Joint Scientific Meeting of the Philadelphia County Dental Society hosted by the U.S. Naval Dental Officers in the FOURTH Naval District will be held at the Naval Hospital, Philadelphia, Wednesday, 19 April 1967, at eight o'clock. Roger G. Gerry DMD, Chief, Dental and Oral Surgery Service, Mount Sinai Hospital Services, City Hospital Center at Elmhurst, N.Y., will present "*Total Extraction for Immediate Dentures Under Local Anesthesia Supplemented with Intra-Venous Tranquilizers*". Dr. Gerry is on the Retired List of the U.S. Navy in the rank of Captain, having served on continuous active duty from 1941 to 1965. —Commandant, FOURTH Naval District, Phila., Pa.

SV-2 SURVIVAL VEST

In response to sea survival needs and a pilot overweight and center of gravity problem due to mislocation of needed equipment, a new item, the SV-2 Survival Vest has been developed at the Aerospace Crew Equipment Laboratory for use by pilots and aircrewmen of fighter/attack aircraft. The vest

contains items that help locate the downed aircrewmen such as radio, signal flares and launcher, distress marker light, mirror and whistle, and also houses a shroud line cutter, a survival knife, gun, and ammunition, as well as the Survival, Escape and Evasion Kit (SEEK-2). The SEEK-2 Kit contains medical aids, rations, and a myriad of specialized equipment to aid long-term land survival. Manufacture of the new vest is described in Clothing and Survival Equipment Change No. 26 dated 8 August 1966.—Public Affairs Office, BuMed.

LARGEST CLASS EVER

The *largest class ever* graduated from the School of Submarine Medicine's rigorous six-month course at New London, Conn. The 40 medical officers will soon report to operating units of the Polaris Submarine Fleet.—Commanders Digest, January 11, 1967.

ACKNOWLEDGMENT

In the U.S. Navy Medical News Letter 49(3):10 February 1967 on page 28, right column, the 5th line should read ". . . Section, Perth, W.A." instead of W. Va.

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